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PATHOLOGY OF SO-CALLED "ACUTE PYELITIS" IN INFANTS *

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Fever and the presence of moderate or large amounts of pus in the urine of infants or young children usually lead to the diagnosis of pyelitis. Based on these criteria, pyelitis is a common disease, but the frequency with which it is diagnosed clinically is in striking contrast to its extreme rarity at postmortem examination. Infants suffering from so-called pyelitis usually recover, but when death occurs, most often due to some intercurrent disease, inflammation of the pelvis of the kidney is rarely found on postmortem examination. Such experiences have led a number of observers to doubt the dependency of pyuria in the young on inflammation of the pelvis of the kidney, but suggest its origin in some other part of the urinary tract (Thiemich,[1] Bugbee [2] and Chown [3]).

During the past twelve years, we have had an opportunity to study postmortem material from forty-nine infants and young children whose urine contained pus during life. A study of this material demonstrates, we believe, that the usual cause of so-called pyelitis is a true suppurative lesion of the interstitial tissue of the kidney itself. The patients included in this study were under 3 years of age and the disease was of the variety usually termed "acute pyelitis." Our results and conclusions are therefore restricted to this type of case.

Chronic pyuria affecting particularly older children is frequently associated with some congenital obstruction in the urinary tract which, in the opinion of many, leads to dilatation and infection of the ureters

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1. Thiemich, M.: Ueber die eitrigen Erkrankung der Nieren und Harnwege im Säuglingsalter, Jahrb. f. Kinderh. **72**:243, 1910.

2. Bugbee, H. G.: Infections of the Urinary Tract in Children, N. Y. State J. Med. **25**:1063 (Dec. 1) 1925.

3. Chown, B.: Pyelitis in Infancy, Arch. Dis. Child. **2**:97 (April) 1927.

and pelvis of the kidney with secondary involvement of the kidney itself (Brewer [4], Hunner [5], Abt [6], Helmholz [7], Bugbee [8], Campbell and Lyttle [9] and others). We have observed such cases but, in our opinion, they present as a rule a different problem from so-called "acute pyelitis."

Our patients presented varying degrees of pyuria.[10] In the milder forms the urine contained moderate amounts of pus during the course of some acute infection. The severer cases were represented by infants who were acutely ill with prostration, high fever and marked pyuria; when other infections were present, they seemed relatively insignificant and insufficient to account completely for the severe symptoms. This group represents the severer type of so-called "acute pyelitis." Our observations indicate, however, that there is merely a difference in degree between the severe cases in which the clincal picture is dominated by the pyuria and the milder ones in which pyuria is apparently an incident or complication of some readily apparent acute infection.

From study of the postmortem material at our disposal, we found that the most common pathologic changes in so-called acute pyelitis of infants were inflammatory foci in the interstitial tissue of the kidney. In their earliest and simplest form, the foci are small circumscribed areas of infiltration by small mononucleated and polymorphonuclear cells frequently adjacent to small blood vessels. As the disease

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4. Brewer, G. E.: The Present State of Our Knowledge of Acute Renal Infection, J. A. M. A. **57**:179 (July 15) 1911.

5. Hunner, G. S.: Urethral Stricture and Chronic Pyelitis in Children, Am. J. Dis. Child. **34**:603 (Oct.) 1927.

6. Abt. I. A.: Pyuria in Malformations of the Urinary Tract, Am. J. Dis. Child. **32**:507 (Oct.) 1926.

7. Helmholz, H. F.: Congenital Anomalies of the Urinary Tract in Childhood, J. A. M. A. **89**:1932 (Dec. 3) 1927.

8. Bugbee, H. G., and Wollstein, M.: Surgical Pathology of the Urinary Tract in Infants, J. A. M. A. **83**:1887 (Dec. 13) 1924.

9. Campbell, M. F., and Lyttle, J. D.: Ureteral Obstruction in Infancy: A Clinico-Pathologic Study of Seventy-Four Cases, J. A. M. A. **92**:544 (Feb. 16) 1929.

10. We have used the term pyuria to designate the presence of sufficient pus to cause definite cloudiness of the urine. We have further restricted the term to cases in which the pus cells were not only very numerous on microscopic examination, but were present in definite clumps. Specimens from female infants were collected so as to exclude contamination with vaginal discharge, which is a frequent source of pus cells in the urine and therefore likely to cause confusion. In a few cases (numbers 6 and 8, table 1), the urine was not examined during life but the diagnosis was based on the presence of pus in the bladder at postmortem examination or the occurrence of pus cells in the urinary tubules revealed by microscopic examination of the kidney.

advances or is more severe, the lesions are larger and contain more polymorphonuclear cells until in the severest forms frank abscess formation is evident, presenting the typical picture of acute suppurative nephritis. The smaller lesions present in the early or milder cases or representing the beginning of the more severe form of the disease are not accompanied by necrosis. They cause only slight destruction of adjacent kidney tissue and probably heal without the formation of scars. More advanced lesions lead to varying degrees of necrosis and in healing are replaced by fibrous tissue.

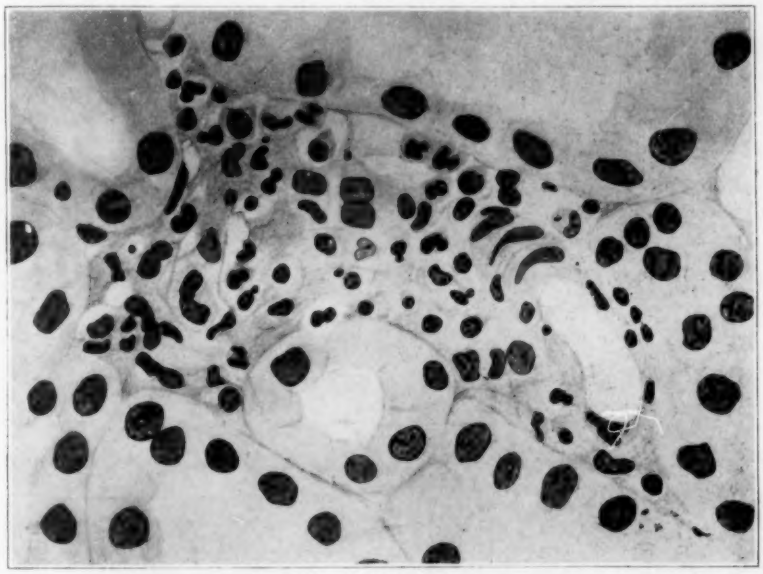

Fig. 1.—Early acute inflammatory lesion of the interstitial tissue of the kidney in the immediate neighborhood of a small blood vessel; high power magnification.

In a recent paper, Chown [3] reviewed the literature and presented observations which led to conclusions identical with our own. He pointed out the lack of evidence favoring the occurrence of true pyelitis in infancy and emphasized the importance of suppurative nephritis as a frequent cause of pyuria in early life.

For purposes of clarity, we believe that our material can be presented best by describing cases which illustrate different phases of the disease and the accompanying lesions of the kidney.

REPORT OF CASES

CASE 1.—*Development of pyuria during the course of severe diarrhea in a boy, aged 6 months. Death on the fourteenth day of the disease. Focal suppurative lesions in the kidney at necropsy.*

A. I., a boy, aged 6 months, was admitted to the hospital in November, 1916, suffering from diarrhea and vomiting of four days' duration. The temperature was 103 F. on admission and ranged from 101 to 104 F. during the following ten days. Severe diarrhea and dehydration were the most pronounced symptoms. Otitis media developed three days after admission. The urine was normal on admission, but four days later it contained a moderate number of pus cells and a faint trace of albumin. The amount of pus increased somewhat, and at the time of death on the tenth day the urine showed a considerable number of free and clumped pus cells, a trace of albumin and a few granular casts.

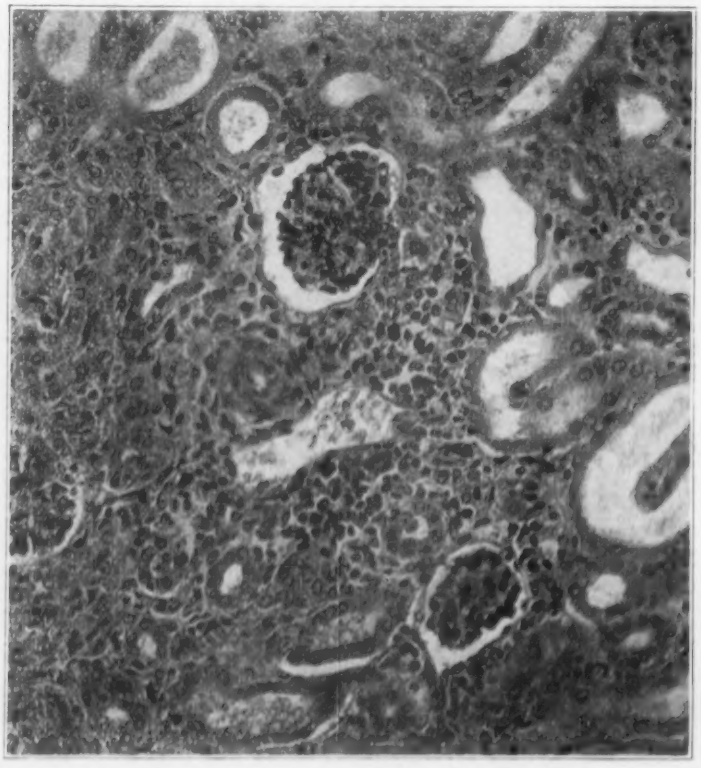

Fig. 2.—An early acute inflammatory lesion near a small blood vessel.

Necropsy disclosed marked malnutrition, otitis media and beginning bronchopneumonia. With the exception of slight congestion of the kidneys, the urinary tract was normal on gross examination. On microscopic examination the kidneys contained small scattered areas of infiltration by round and polymorphonuclear cells. Such foci were confined to the interstitial tissue, frequently near the small blood vessels. Definite necrosis was not observed, but frequently tubules adjacent to the foci were encroached on and destroyed.

Sections from the pelves of the kidneys, ureters and bladder showed no inflammatory reaction.

Various acute infections, especially those of the upper respiratory tract, are not infrequently complicated by so-called pyelitis.[11] Most of these patients recover, and the pathologic anatomy of the lesion is never known. It is possible, however, when pyuria complicates a fatal illness, to attempt to determine its origin by postmortem examination of the urinary tract. Case 1 is one of a series of twenty-eight in which we were able to carry out such a study. In only one case (number 8, table 1) was there any definite lesion of the pelvis of the kidney. Minute focal accumulations of leukocytes were observed in

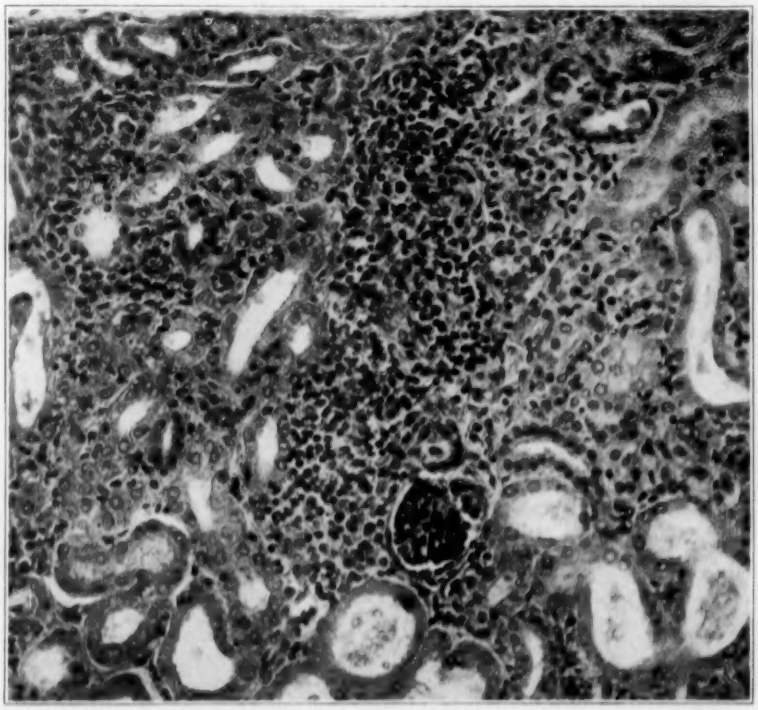

Fig. 3.—An early inflammatory lesion situated in the cortex just beneath the capsule. There is some destruction of the parenchyma.

the submucosa and there was one similar area of infiltration of the lining epithelium. In the interstitial tissue of both kidneys, however, there were focal suppurative lesions which were much more marked than those in the renal pelvis.

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11. The frequency with which "so-called pyelitis" follows other infections in the body is suggestive. It seems possible that bacteria may be transported directly to the kidney and excite there an acute inflammatory lesion such as is known to occur in septicemia. This relationship, however, is suggestive only and cannot be considered direct unless the same variety of micro-organism is demonstrated as the cause of both lesions.

TABLE 1.—*Cases of Mild or Moderate Pyuria Complicating Acute Infections; Showing the Absence of Pyelitis and the Presence of Focal Suppurative Lesions in the Kidney*

No.	Age, Mos.	Sex	Diagnosis	Pyuria	Urinary Tract at Postmortem Examination
1	2¼	F	Pyuria; diarrhea; intestinal intoxication; otitis media; cellulitis of trunk	++	Macroscopically, left kidney normal; minute gray dots and short streaks in right kidney; right kidney considerably swollen; microscopically, both kidneys show small focal acute suppurative lesions and some have reached the stage of miliary abscesses; remainder of urinary tract normal
2	16	M	Pyuria; septicemia	++	Both kidneys swollen; many minute red dots in both cortex and medulla; sections show small focal areas of acute inflammation and miliary abscesses; remainder of urinary tract normal
3	6	F	Pyuria; acute meningitis; otitis media	+	Macroscopically, urinary tract normal; minute focal interstitial areas of inflammation in both kidneys; remainder of urinary tract normal
4	8½	M	Pyuria; rickets; tetany; pneumonia	+	Mild acute inflammation of lining of bladder; no lesions in kidneys, pelves or ureters
5	7	F	Pyuria; abscesses of lungs; otitis media	+	No lesion of urinary tract
6	2¼	M	Pyuria; diarrhea; intestinal intoxication; otitis media; pneumonia	++	Urinary tract grossly normal; numerous small foci of acute inflammation in kidneys; remainder of urinary tract normal
7	2	M	Pyuria; diarrhea; intestinal intoxication; otitis media	++	A few scattered bright red dots at lower pole of left kidney; moderately dilated left pelvis and ureter; both kidneys show numerous focal areas of acute inflammation and a few miliary abscesses; pelves and bladder show a few mononucleated cells in the subepithelial tissue; ureters normal
8	1	F	Pyuria; septicemia (?); pneumonia	++	Minute focal acute inflammatory areas in the submucosa of the pelves and, in one instance, in the lining epithelium; focal acute suppurative lesions of the kidneys were present and of more marked degree
9	1½	M	Pyuria; diarrhea; intestinal intoxication; otitis media	+	Urinary tract grossly normal; small focal areas of inflammation in kidneys; remainder of urinary tract normal
10	11	F	Pyuria; diarrhea; intestinal intoxication; pneumonia; otitis media	++	Urinary tract grossly normal; small focal areas of inflammation in kidneys; remainder of urinary tract normal
11	2¼	M	Pyuria; diarrhea; intestinal intoxication; parotid abscess; otitis media	+	Kidneys grossly normal; ureters show "U" shaped kinks at brim of pelvis but no apparent diminution in caliber; small focal areas of inflammation in both kidneys; remainder of tract shows no evidence of inflammation
12	7	F	Pyuria; acute meningitis (pneumococcus); otitis media	++	Both kidneys show many focal areas of inflammation, some of which are miliary abscesses; remainder of urinary tract normal
13	4½	F	Pyuria; congenital syphilis; pneumonia	++	Both kidneys show scattered focal areas of inflammation and a few miliary abscesses; remainder of urinary tract normal
14	6	F	Pyuria; acute meningitis (meningococcus); acute vaginitis	+	Kidneys grossly normal; scattered focal areas of inflammation in both kidneys; a few lymphoid and plasma cells in the subepithelial tissue of the pelves; remainder of urinary tract normal
15	3	F	Pyuria; acute infectious enteritis; otitis media	++	Numerous small grayish areas in cortex and medulla; numerous focal areas of inflammation and miliary abscesses in both kidneys; slight perivascular lymphoid and plasma cell reaction in subepithelial tissue of pelves and bladder; urinary tract otherwise normal
16	7	M	Pyuria; pneumonia; otitis media	++	Slight congestion of bladder; ureters and pelves of kidneys negative; small suppurative lesions in interstitial tissue of kidney

TABLE 1.—*Cases of Mild or Moderate Pyuria Complicating Acute Infections; Showing the Absence of Pyelitis and the Presence of Focal Suppurative Lesions in the Kidney—Continued*

No.	Age, Mos.	Sex	Diagnosis	Pyuria	Urinary Tract at Postmortem Examination
17	7½	M	Pyuria; diarrhea; intestinal intoxication; bronchopneumonia	++	Focal inflammatory lesions in interstitial tissue of kidney; ureters, bladder and pelves of kidney normal
18	6½	F	Pyuria; diarrhea; otitis media; intestinal intoxication	++	Focal areas of inflammation in interstitial tissue of kidney; ureters, bladder and pelves of kidney normal
19	8½	M	Pyuria; bronchopneumonia	++	No lesions in urinary tract
20	12	F	Pyuria; bronchopneumonia; empyema	++	Very few focal inflammatory areas in interstitial tissue of kidney; remainder of urinary tract normal
21	9	F	Pyuria; furunculosis; malnutrition; empyema	++	Foci of acute inflammation in interstitial tissue of kidney; remainder of urinary tract normal
22	14	F	Pyuria; otitis media; meningitis	++	No lesions of urinary tract
23	11	M	Pyuria; bronchopneumonia; acute peritonitis	++	Focal areas of inflammation in interstitial tissue of kidney; remainder of urinary tract normal
24	20	F	Pyuria; colitis; furunculosis	++	Ureters and bladder normal; small areas of infiltration in both kidneys; few areas of infiltration in subepithelial layers of pelvis of kidney
25	26	F	Pyuria; epidemic cerebrospinal meningitis	++	No significant lesions of urinary tract
26	30	F	Pyuria; bronchopneumonia; otitis media	++	Ureters, pelves of kidneys and bladder normal; focal inflammatory lesions in interstitial tissue of both kidneys
27	37	F	Pyuria; lobar pneumonia	++	No significant lesions of urinary tract
28	40	M	Pyuria; otigenic meningitis	++	Focal areas of inflammation in interstitial tissue of kidney; remainder of urinary tract normal

In twenty-two of the twenty-eight cases of this group (table 1), the kidneys showed microscopic lesions similar to those described.[12]

In six cases no lesions were found in the kidney, but in one of these (number 4, table 1) a mild cystitis seemed sufficient to account for the pyuria present during life. In the remaining five cases, however, no anatomic basis for pyuria was disclosed on postmortem examination. It is possible that these cases belong to the group in which pyuria is ascribed to a catarrhal inflammation of the urinary tract (Finkelstein, Rosenstein and others).

To accept this explanation, however, would necessitate the assumption that a grade of inflammation existed during life sufficient to cause the appearance of pus in the urine with the total disappearance of such lesions after death. Whether this assumption is true or false seems too indefinite to merit detailed discussion.

12. In fifteen cases, sections of the kidneys stained by the Giemsa method were examined to determine the presence of bacteria in the inflammatory foci. In eleven cases, no bacteria were found. The lesions contained bacilli in three cases and cocci in one case.

The frequency of suppurative lesions of the kidneys in this group of patients and the absence of other significant pathologic changes in the urinary tract would seem to justify the belief that interstitial suppurative nephritis is the cause of pyuria in the majority of such cases.

TABLE 2.—*Cases of Marked Pyuria Accompanied by High Fever and Prostration; Suppurative Nephritis Found at Necropsy*

No.	Age, Mos.	Sex	Diagnosis	Pyuria	Urinary Tract at Postmortem Examination
1	5½	F	Pyuria; otitis media	+++	Extensive acute suppurative lesions of both kidneys with abscess formation; pelves of kidneys normal
2	8	F	Pyuria; lobar pneumonia	+++	Acute suppurative lesions of kidneys; remainder of urinary tract grossly normal
3	¾	M	Pyuria; mild acute infection of upper respiratory tract	++++	Numerous acute suppurative lesions in both kidneys; small accumulations of lymphoid and plasma cells in subepithelial tissue of pelves of kidneys; two polypoid growths in wall of bladder; urinary tract otherwise normal
4	12	M	Pyuria; rickets; anemia (secondary)	+++	Acute suppurative lesions of kidneys with abscess formation; scattered collections of lymphoid and plasma cells in subepithelial tissue of pelves of kidneys; remainder of urinary tract grossly normal
5	4½	M	Pyuria	+++	Scattered, irregularly distributed acute suppurative lesions of the kidneys; an occasional polymorphonuclear in the pelvic mucosa; remainder of urinary tract normal
6	7	F	Pyuria; otitis media	++++	Irregular areas of acute suppurative inflammation in both kidneys without definite abscess formation; occasional perivascular accumulations of lymphoid and plasma cells in subepithelial tissue of pelves of the kidneys; remainder of urinary tract normal
7	2	F	Pyuria; pneumonia; otitis media	+++	Acute suppurative lesions of kidneys with small abscesses; scattered lymphoid and plasma cells in pelvic mucosa; remainder of urinary tract normal
8	¼	F	Pyuria; recto-vaginal fistula	+++	Widely scattered acute suppurative lesions of the kidneys with small abscesses; dilatation of both ureters; pelves, ureters and bladder show no suppurative lesions
9	17	M	Pyuria; multiple abscesses of lungs; empyema	++++	Acute suppurative lesions of the kidneys; remainder of urinary tract normal
10	⅔	M	Pyuria; pneumonia	+++	Acute suppurative lesions of the kidneys with abscess formation; remainder of urinary tract normal
11	4	M	Pyuria	+++	Extensive acute suppurative inflammatory processes in pelves of kidneys; extensive acute suppurative lesions of the kidneys with abscess formation; the lesions of the kidneys were of more marked degree than the lesions of the pelves
12	½	M	Pyuria; acute meningitis (B. coli); peritonitis (B. coli)	++++	Acute suppurative lesions of the kidneys; a few focal accumulations of lymphoid and plasma cells in the subepithelial pelvic and ureteral tissue; urinary tract otherwise normal
13	7	M	Pyuria; otitis media	+++	Multiple acute suppurative lesions of both kidneys; in some areas frank abscess formation
14	8	M	Pyuria; retro-pharyngeal abscess; tonsillitis	++++	Multiple acute suppurative lesions of both kidneys, few definite abscesses
15	8	F	Pyuria; broncho-pneumonia	++++	Multiple abscesses of both kidneys
16	11	F	Pyuria; otitis media; severe diarrhea	+++	Numerous small abscesses in various stages of development
17	13	M	Pyuria; colitis	++++	Numerous small abscesses in varous stages of development

CASE 2.—*Marked pyuria in a girl, aged 7 months. Extensive suppurative lesions of the kidneys found on postmortem examination.*

P. M. L., a white girl, aged 7 months, was admitted to the hospital on March 4, 1923. She had been ill about ten days with fever and drowsiness. The temperature was 104 F. on admission; there was slight stiffness of the neck and both ear drums were red and bulging. Paracentesis of the ear drums revealed pus in the left middle ear. The urine contained large numbers of pus cells, both free and in clumps, a trace of albumin and a few granular casts. At the time of admission, the kidneys were not palpable, but the left could be felt three days

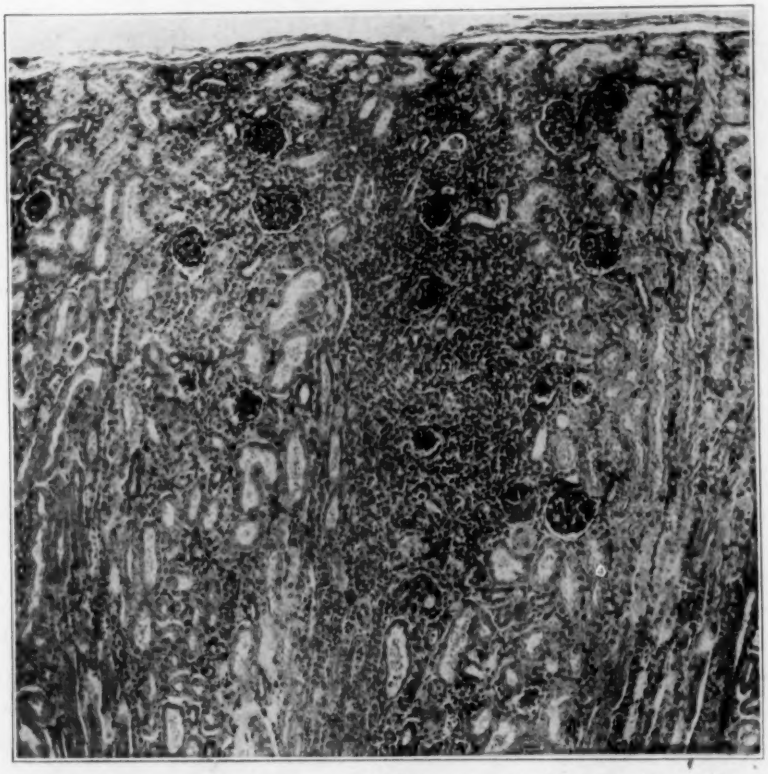

Fig. 4.—A more advanced acute inflammatory lesion showing infiltrating leukocytes and destruction of the renal tubules.

before death. The temperature ranged from 101 to 106 F. Two blood cultures were sterile.

Postmortem examination showed bilateral suppurative otitis media and suppurative nephritis; the other organs were normal. Both kidneys were enlarged, the left more than the right. Both contained many irregular gray areas scattered throughout their substance. On microscopic examination these areas were found to be definite abscesses in various stages of development. The intervening kidney tissue appeared normal.

The pelves of the kidneys, the ureters and the bladder were normal on gross examination. On microscopic examination, there were small perivascular accumulations of lymphoid cells in the deeper layers of the renal pelvis; in other respects the urinary tract below the kidney was normal.

This case is representative of seventeen similar cases (table 2), and in only one of these was there definite inflammation of the pelvis of the kidney. This, however, was insignificant in comparison with the more severe and more prominent lesions in the renal tissue.

Postmortem examination showed clearly that in this group of cases pyuria was due to suppurative nephritis, but the clinical picture was indistinguishable from that of so-called "acute pyelitis." Had any of these patients recovered, the diagnosis of pyelitis would have been made, notwithstanding the fact that in similar cases postmortem examination usually fails to reveal any pronounced lesion of the renal pelvis but practically always acute suppurative renal lesions. Despite the results of postmortem examination, it may perhaps be assumed that pyelitis is the primary disease with secondary involvement of the kidney. It seems difficult, however, to reconcile this view with the absence of inflammation of the renal pelvis at necropsy. Most patients recover from so-called "acute pyelitis," and perhaps the reason for the persistence of the conception of pyelitis as the usual cause of severe pyuria is that suppurative nephritis is not generally regarded as a disease from which spontaneous recovery is frequent. On the other hand, spontaneous recovery seems more consistent with inflammation of the renal pelvis owing to greater opportunities for drainage. Such reasoning, however, does not seen in accord with clinical observation, and it seems probable that spontaneous recovery from severe suppurative nephritis is not infrequent. Case 3 is given in illustration.

CASE 3.—*Marked pyuria in a boy, aged 4 months. Multiple abscesses in one kidney removed at operation. Subsequent enlargement of remaining kidney. Recovery.*

S. E., a white boy, aged 4 months, was admitted to the hospital on Sept. 15, 1922, with a high temperature, ranging from 102 to 105 F.; the urine contained considerable pus.

The left kidney was palpable, and a diagnosis of suppurative nephritis was made. Owing to the enlargement of the left kidney only and the severity of the symptoms, operation was decided on. The left kidney was removed and showed an acute suppurative nephritis and a large number of small abscesses. The mucous membrane of the pelvis of the kidney appeared normal, but, on later microscopic examination, a slight lymphoid and plasma cell infiltration was found in the subepithelial tissue.

Despite the removal of the kidney, fever, severe general symptoms and pyuria continued, and two days after operation the right kidney became enlarged. The symptoms, although improved, persisted for the following ten days. At the end of this time the infant was taken home by the parents, and the symptoms gradually abated and then disappeared. The infant was followed in the outpatient department, and one year after the illness was free from pyuria and seemed entirely well.

The clinical history of this patient is fairly typical of so-called "acute pyelitis." An accurate diagnosis was possible only by examination of the removed kidney. The persistence of the pyuria and general

symptoms following operation with enlargement of the remaining kidney strongly suggests that the lesions in both kidneys were identical.

This case demonstrates spontaneous recovery from severe suppurative nephritis and supports similar observations by Forbes [13], Chown [3], Bugbee [2] and others. Further evidence of the true nature of acute pyelitis is furnished by three other cases in which a postmortem examination some time after the acute disease showed healed lesions. One case will be cited.

CASE 4.—*Marked pyuria in a girl, aged 2 years. Duration of illness about six weeks. Postmortem examination two years later showing evidence of healed suppurative nephritis.*

A. I., a girl, aged 2 years, was admitted to the hospital on Jan. 3, 1916. The child had been ill for two weeks with fever, slight cough, restlessness and anorexia.

TABLE 3.—*Cases of Pyuria Followed by Recovery; Subsequent Infectious Diseases Outside the Urinary Tract Resulting in Death; Healed Lesions of the Kidneys Found at Necropsy*

No.	Sex	Age	Age When First Showed Pyuria, Years	Principal Cause of Death	Pyuria	Urinary Tract at Postmortem Examination
1	F	4 years	2	Pneumonia	..	Scattered areas of scar tissue in both kidneys varying from 2 to 5 mm.; pelves, ureters and bladder showed no evidence of old or recent inflammatory processes
2	F	5 years, 3 mo.	1½	Acute myocarditis and endocarditis with thrombus formation; pyuria	..	Irregular areas of scar tissue most numerous in the lower pole of the left kidney; scattered focal areas of acute inflammation in both kidneys; pelves, ureters and bladder without evident lesion
3	M	3 mo.	?	Pneumonia	..	Scattered areas of scar tissue in both kidneys; pelves, ureters and bladder without evident lesion

Except for slight congestion of the tonsils and pharynx, the results of the routine physical examination were negative. The urine was cloudy and contained great numbers of pus cells and a moderate amount of albumin.

The patient was in the hospital five weeks, during which time the temperature ranged from 101 to 106 F. The fever gradually subsided and the child was discharged with normal temperature, although the urine still contained a few pus cells. When seen two months later, the child seemed well and the urine was entirely free from pus.

About two years later, the child died from pneumonia after a residence of three days in the hospital.

Postmortem examination showed scattered areas of scar tissue in both kidneys, especially in the cortex. Such areas varied from 2 to 5 mm. in their largest dimension and were irregular in outline. It seemed probable that these scars were the result of previous abscess formation. This diagnosis was confirmed by histologic examination. No other renal changes were discoverable.

13. Forbes, J. G.: Infections of the Urinary Tract with B. Coli in Infancy and Childhood, Brit. M. J. **2**:1133 (Oct. 15) 1910.

We have observed two other cases similar to the one described. One was of special interest. The patient, a girl, aged 5 years, had had a previous attack of severe pyelitis, from which she had recovered. She was admitted to the hospital in February, 1927, suffering from acute myocarditis, and again developed pyuria. At postmortem examination, the kidneys showed multiple scars probably due to the previous "pyelitis" and fresh active lesions associated with the recent pyuria.

COMMENT

The cases which have been cited are representative of the lesions found at postmortem examination of forty-nine infants and young children whose urine contained pus during life. They have in no

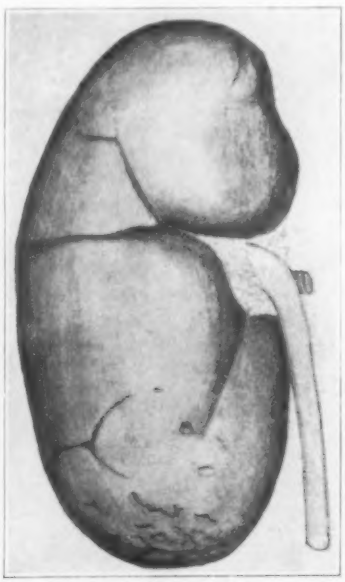

Fig. 5.—Scarring of the kidney following attacks of so-called acute pyelitis.

instance afforded justification for the diagnosis of pyelitis. The epithelial surface of the pelvis of the kidney was inflamed in only two cases (no. 8, table 1 and no. 11, table 2), but in one (no. 8, table 1) the inflammation was of limited extent and degree. Minute areas of round cell infiltration of the subepithelial layers of the pelvis of the kidney were found on histologic examination of a few cases (nos. 7, 15 and 24, table 1 and nos. 3, 6, 7 and 12, table 2). These lesions were unimpressive as a cause of pyuria since in addition to their infrequency and the small areas which they involved, they were accompanied always by more striking and more significant lesions of the kidney itself. A review of the literature also yields no evidence in favor of the view that

pyelitis is a common cause of pyuria in infants. The only exception is the case reported by Cabot and Crabtree.[14] It seems unnecessary for us to review the literature in detail, as a comprehensive review is contained in the excellent paper of Chown.[3]

In contrast to the absence of pyelitis, we have found in most of our cases definite focal inflammatory lesions in the interstitial tissue of the kidney. The larger and more marked lesions which were found in the severer cases were acute suppurative lesions frequently showing small abscesses and would therefore seem unlikely to provoke discussion as a cause of pyuria. In the milder cases of pyuria the lesions were smaller, less conspicuous and frequently consisted largely of lymphocytes and plasma cells, although polymorphonuclear cells were always present in considerable numbers. Owing to their small size, some doubt might arise concerning the significance of such lesions. We therefore studied the kidneys of infants whose urine, during life, did not contain pus. In a few such cases, small accumulations of lymphoid cells were found in the interstitial tissue of the kidney similar to those normally present in other organs, but these did not appear to be the result of inflammation nor did they encroach on or destroy adjacent parenchyma, as was true of the lesions we consider significant. A further distinction of importance is the presence of at least moderate numbers of polymorphonuclear cells in the truly inflammatory lesion. It is also of significance that the smaller and less conspicuous lesions were frequently found in association with larger unmistakable abscesses, and definite gradations between the two were often present in the same kidney. We believe, therefore, that the smaller foci which were found in the milder cases of so-called pyelitis are not incidental, but in all probability represent the pathologic lesion which gives rise to pyuria.

It is not our opinion that pyuria in infants is always due to suppurative interstitial nephritis. On the contrary, there are a number of other conditions to which it may be due, but in our experience these are relatively rare. Congenital anomalies of the urinary tract, especially those leading to dilatation of the ureters and pelves of the kidneys, are often associated with chronic inflammation of the mucous membrane of these structures. Cystitis occasionally occurs in infants. Renal calculi, while rare, may provoke pyelitis in early life.

It is our belief, however, based on the evidence set forth, that the most common cause of severe acute pyuria in young infants, especially the type of case usually designated pyelitis, is an acute inflammatory process of the interstitial tissue of the kidney.

There has been considerable discussion in the pediatric literature concerning the origin of pyelitis, whether it represents an infection

14. Cabot, H., and Crabtree, E. G.: The Etiology and Pathology of Non-Tuberculous Renal Infections, Surg. Gynec. Obst. **23**:495 (Nov.) 1916.

which is hematogenous or ascending. The main point of evidence in favor of the latter view is the greater frequency of the disease in females. There is perhaps some question as to whether this sex difference actually exists to the degree to which it has been supposed, since neither in our series of cases nor in that of Chown [3] was the predominance of females as striking as is usually supposed. Aside from this, however, inflammatory foci in the interstitial tissue of the kidney in the absence of inflammation of the bladder, ureters or pelves of the kidneys would seem to indicate a blood-borne infection rather than one which had ascended through the entire urinary tract without causing lesions. Brewer,[4] some years ago, on the basis of both experimental and clinical observation, pointed out that ascending infections of the kidneys are always the result of obstruction and stasis. In such cases, the inflammation was not restricted to the tissue of the kidney, but involved also the ureters and the pelvis of the kidney.

We have nothing to constribute to therapy. In view of our observations it seems evident why the usual medical methods of treating so-called "acute pyelitis" have been of such little value. When a severe suppurative process affects only one kidney, the question of its removal must give rise to serious consideration.

SOLITARY NONPARASITIC CYSTS OF THE LIVER*

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Solitary nonparasitic cysts of the liver occur with sufficient rarity to make this report of two cases of interest. In 1913, Boyd [1] collected thirty-four such cases reported in the medical literature, operation having been performed in each case. In 1923, John F. X. Jones [2] of Philadelphia observed another instance, and in an excellent review of the literature on the subject, found that the reported number of cases in which operation had been performed at that time aggregated sixty-one. Jones quoted from a letter from Stuart Harrington of the Mayo Clinic, dated July 18, 1922, in which it was stated that twenty-five cases of solitary cyst of the liver had been observed and operation performed at the Mayo Clinic. In 1926, Harrington [3] reported the successful removal of a multilocular hemorrhagic cyst of the liver. Orr and Thurston [4] added another case in 1927, and collected thirteen other instances in which operation had been performed since the report of Jones. In 1928, Faltin [5] reported an instance in which four years before he had successfully removed a large nonparasitic cyst of the liver.

To this total of 102 cases of nonparasitic cysts of the liver, in which operation has been performed, we wish to add two of our own. One of these, because of the size of the cyst and the age of the patient (the youngest among the series), is of unusual interest.

REPORT OF CASES

CASE 1.—M. R., a boy, aged 10 months, was admitted to the University Hospital on Sept. 17, 1928. At the age of 7 months, the mother noticed an abdominal swelling, and this had gradually increased. There had been no pain or gastrointestinal disturbance. The child was born at full term by normal delivery. The birth weight was 7¾ pounds (3,515.34 Gm.), and the gain in weight was satisfac-

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1. Boyd, Sidney: Non-Parasitic Cysts of the Liver, Lancet **1**:951 (April 5) 1913.

2. Jones, J. F. X.: Retention Cyst of Liver, Ann. Surg. **77**:68, 1923.

3. Harrington, S. W.: Solitary Non-Parasitic Cyst of Liver, S. Clin. N. Amer. **6**:1191 (Oct.) 1926.

4. Orr, T. G., and Thurston, J. A.: Strangulated Non-Parasitic Cyst of the Liver, Ann. Surg. **86**:901, 1927.

5. Faltin, R.: Lebercyste, Acta Chir. Scandinav. **64**:375, 1928.

tory, the child being nursed for seve months, after which he received whole milk, cereal and vegetables. The family history was essentially negative.

Physical Examination.—Examination revealed a fairly well nourished white boy, 10 months of age. The abdomen was protuberant, and on palpation, it was found to be almost filled with a nontender mass. There was definite bulging in both flanks. The outline of the mass could not be definitely established. The mass, though rather firm, imparted the feel of a moderately tense cyst. The greater portion of the abdomen gave a dull note on percussion. Only in the lower left quadrant was tympany demonstrated. The mass did not move with respiration.

Fig. 1 (case 1).—M. R., aged 10 months, before operation. Note the prominent abdomen due to the large hepatic cyst.

The chest and heart were normal on examination. There was a slight rhinitis; the remainder of the examination gave negative results.

Repeated laboratory examinations revealed a normal urine and a normal blood picture; the Wassermann reaction of the blood and the Pirquet test were negative. A flat plate of the abdomen demonstrated a large mass on the right side, extending well over to the left and displacing the intestinal coils downward and to the left.

Preoperative diagnosis could not be made as the condition was indeterminate, a mesenteric cyst probably being present.

Operation.—One week after admission, the patient was operated on (O. H. W.), a long incision being made over the right rectus muscle. Just before the operation

was begun, 2 minims (0.123 cc.) of surgical pi' itary extract was given subcutaneously. On incising the peritoneum, a large cyst was observed. It was so large that its limits could not be defined until puncture was performed. About 1.5 liter of greenish-brown fluid was removed by aspiraton with a suction apparatus.

The right lobe of the liver was practically absent, only a small piece of liver tissue becoming visible in the lower portion of the wall of the cyst after puncture and removal of the fluid.

There appeared to be no liver tissue intervening on the right between the diaphragm and the upper margin of the cyst. Its left boundary was formed by the left lobe of the liver, and its wall apparently fused with the peritoneum in the region of the ligamentum teres. The gallbladder was present in the lower margin of the wall of the cyst. The piece of liver on the right looked fairly normal, but no definite duct was made out going to the other extrahepatic bile ducts. The

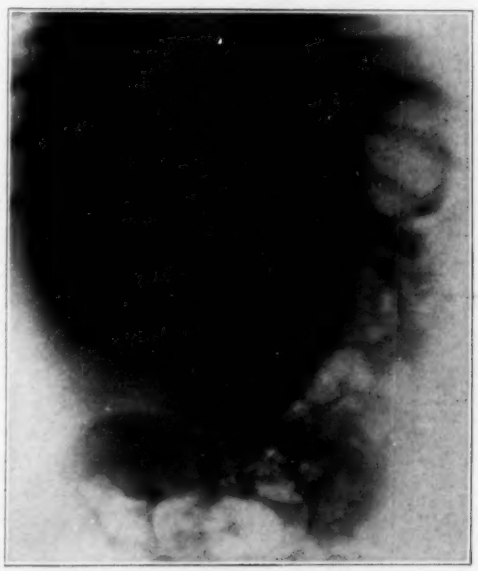

Fig. 2 (case 1).—Roentgenogram revealing a large mass on the right side of the abdomen. The colon is pushed downward and to the left.

normal appearance of this piece of liver tissue, however, indicated that an extrahepatic bile duct must have been present.

Following drainage of the cyst and excision of a good portion of the free border, marsupialization was performed. The wall of the cyst was sutured to the parietal peritoneum, incorporating this portion in the usual closure of the abdominal wound, and a tube left in the cyst for drainage was brought out through the upper portion of the wound.

The specific gravity of the fluid removed at operation was 1.022; 15 Gm. of albumin was present in 1 liter of the fluid; qualitative examinations for bile pigment failed to demonstrate its presence.

Postoperative Progress.—Following operation, the general condition of the patient was good, except that a slight secondary anemia developed; fourteen days after operation, the child had rhinitis, followed by bilateral acute catarrhal otitis media. He recovered from this infection, and was discharged in good health.

There was little drainage from the wound, and aspiration through the tube yielded each day only a few cubic centimeters of blood-tinged fluid, which failed to give the chemical tests for bile pigment. Eleven days following operation, an opaque solution was injected through the tube into the wound, and roentgenograms were taken. They revealed a marked change in the abdomen; the cyst apparently

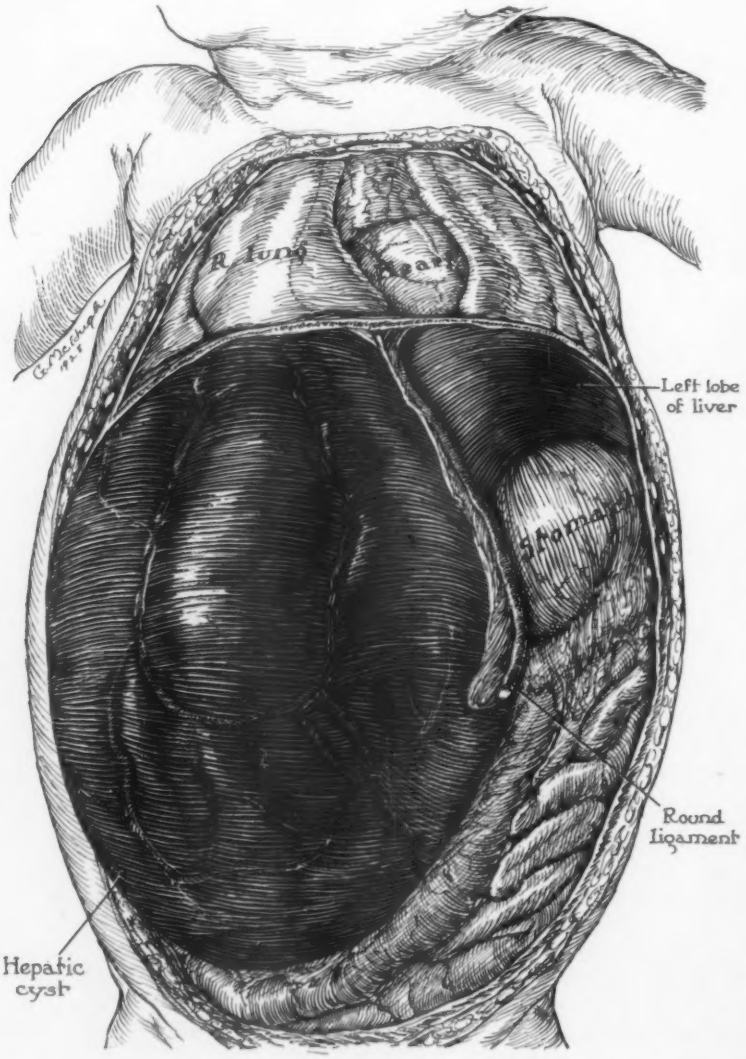

Fig. 3 (case 1).—Sketch showing the position and relations of the cyst as seen on opening the abdomen at the operation.

was already entirely collapsed, and only a few centimeters of fluid could be injected. The descending colon and small bowel previously displaced to the left now filled the right side of the abdomen.

Twenty-two days after operation, the tube came out, there having been scarcely any drainage. Six weeks following operation, another roentgenogram of the abdomen was taken after inflation of the colon with air. The edge of the liver was fairly well outlined, and it was apparent that the right lobe of the liver, though still smaller than normal, had regenerated remarkably.

Microscopic Examination.—The wall of the cyst could be divided histologically into three layers. At some points, this differentiation was fairly distinct; at other places it was not clear. There was no epithelial lining. There was a narrow inner layer of connective tissue which was fairly loose, and which contained large numbers of nuclei, but few blood vessels; then a wide middle layer of loose supporting tissue,

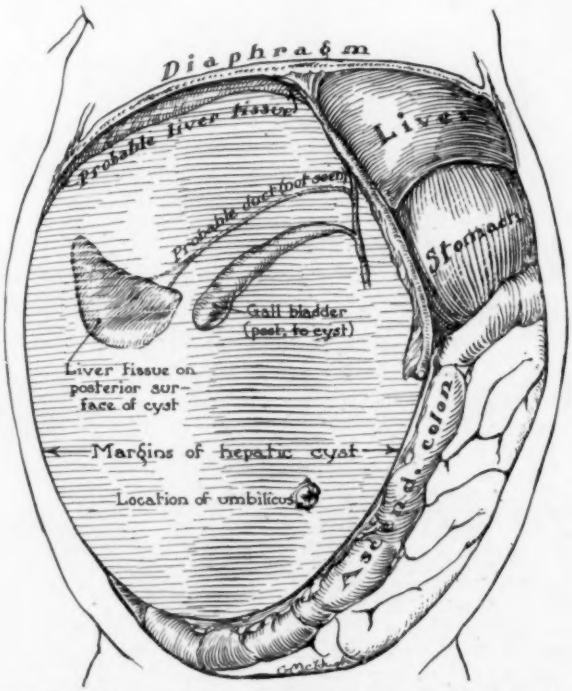

Fig. 4 (case 1).—Diagrammatic sketch showing relation of the small piece of liver tissue to the wall of the cyst on its inferior aspect.

rich in blood vessels of varying sizes. In this layer, there were scattered areas of compressed liver cells, groups of distorted bile ducts and patches of lymphocytes. The outer layer, a little wider than the inner, exhibited a fairly dense stroma and contained a few blood vessels. Many elastic fibers were also present.

The rapidity with which the liver shadow on the right increased in size was remarkable. At operation, it seemed doubtful whether liver tissue was present above the wall of the cyst. This return of the shadow to the usual normal size in six weeks is quite compatible with the well known capacity of the liver to regenerate.

The child was reexamined on February 20; he was in good health, and the wound had healed nicely.

CASE 2.—Mrs. C. J., aged 59, was admitted to the medical service of the University Hospital on Sept. 21, 1925, with the complaint of a mass in the upper part of the abdomen of a year's duration. It had never been tender, nor had she suffered attacks of pain. There had been no quantitative nor qualitative distress from food. The history otherwise was essentially negative.

Physical Examination.—Examination showed a well developed woman, somewhat undernourished, with a small mass in the right hypochondrium, apparently attached to the liver. The edge of the liver extended about 2 cc. below the right

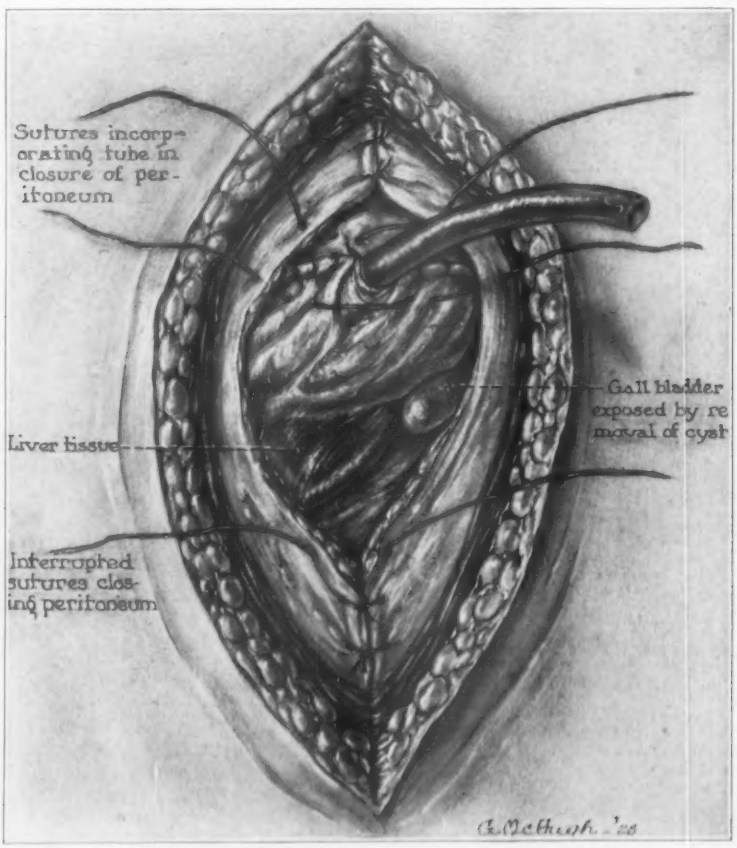
Sutures incorporating tube in closure of peritoneum
Gall bladder exposed by removal of cyst
Liver tissue
Interrupted sutures closing peritoneum

Fig. 5 (case 1).—The method of drainage of cyst and the closure of abdominal wound.

costal margin in the midclavicular line, and the mass rose and descended in the abdomen with the liver on respiration. The mass itself was rounded and seemed about the size of a grapefruit. It was not tender. Other observations were essentially negative. The systolic blood pressure was 148 mm. and the diastolic 110 mm. of mercury.

Laboratory examinations of the urine gave negative results. The blood had a hemoglobin of 60 per cent, with an erythrocyte count of 3,710,000 and a leukocyte count of 7,400. The differential examination of the white cells was normal. The

Wassermann reaction of the blood was negative. The blood sugar was 0.092 and the blood urea 11.2 mg. per hundred cubic centimeters. Roentgen examination of the gastro-intestinal tract revealed no abnormality.

Preoperative diagnosis was probable hydrops of the gallbladder.

Operation.—The patient was transferred to the surgery service and operated on Oct. 8, 1925, under ether anesthesia. Dr. Law, on whose surgical service this patient was operated on, permitted one of us (O. H. W.) to remove the cyst and to include the report of the case in this paper. The operation was performed through an incision high over the right rectus muscle. When the peritoneal cavity was opened, the large mass was found to be a thin-walled cyst, about the size of a large orange, attached to the liver at its lower edge, just to the left of the gall-

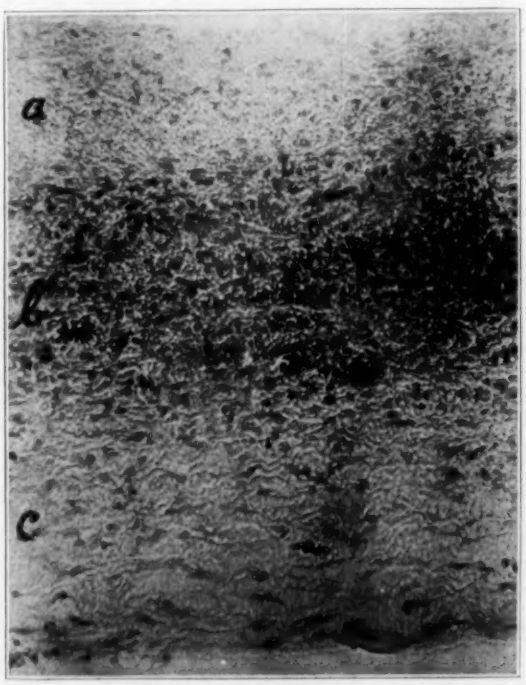

Fig. 6.—Microscopic section of the wall of the cyst, showing: *a*, inner layer of fairly loose connective tissue; *b*, middle layer with many blood vessels and a lymphocytic infiltration, *c*, outer layer of rather dense tissue containing elastic fibers.

bladder. By sharp dissection, the entire cyst was peeled out of the liver bed. The capsular margins of the edges of the liver were resutured. The gallbladder was small and appeared normal. The extrahepatic bile passages were normal. The closure of the abdominal wound was made in layers with catgut and silkworm and linen in the skin.

The fluid in the cyst was clear, and failed to give the chemical tests for bile pigment. Microscopic examination of the wall of the cyst showed it to be devoid of an epithelial lining. Isolated bile ducts were to be seen here and there in the rather loose connective tissue stroma of the wall.

The postoperative convalescence was uneventful, and the patient was discharged as well two weeks after operation.

PATHOLOGY OF NONPARASITIC CYSTS

In 1905, Bland-Sutton [6] divided nonparasitic cysts of the liver into two classes: general cystic disease and solitary cysts. This division has been adhered to by Boyd,[1] and followed in part by Sonntag.[7] Boyd pointed out that in general cystic disease, the liver contains numerous cysts, varying from a microscopic cavity to those as large, or larger, than a pigeon's egg. The cysts may be separated by only a thin layer of tissue. The condition is associated with cystic disease of other organs, especially of the kidneys, but the pancreas, lungs, spleen and brain may

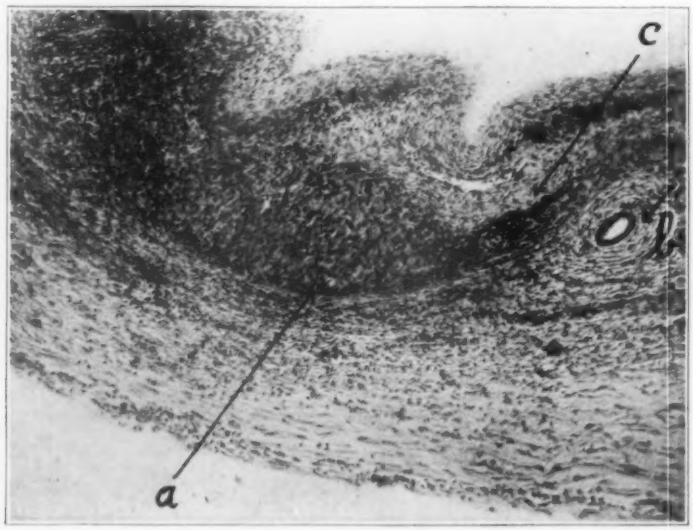

Fig. 7.—Microscopic section of the wall of the cyst showing: *a*, an area of compressed liver cells; *b*, a bile duct, *c*, and patches of lymphocytes in middle layer.

also be involved. Moschcowitz [8] collected eighty-five cases of general cystic disease from the literature, and found that the liver alone was involved in only ten. Shaw and Elting [9] added three more cases, of which two were observed in fetuses. Seven were found in new-born

6. Bland-Sutton, J.: On Solitary (Non-parasitic) Cysts of the Liver, Brit. M. J. **2**:1167, 1905.

7. Sonntag, E.: Beitrag zur Frage der Solitären nicht parasitären Lebercysten, Beitr. z. klin. Chir. **86**:327, 1913.

8. Moschcowitz, E.: Non-Parasitic Cysts (Congenital) of the Liver with a Study of Aberrant Bile Ducts, Am. J. M. Sc. **131**:674, 1906.

9. Shaw, H. L. K., and Elting, A. W.: Congenital Cyst of the Liver with Report of a Case, Arch. Pediat. **26**:818 (Nov.) 1909.

children, one in a child 11 years of age and the remainder in patients over 60. Boyd [1] and Sonntag [7] are inclined to the belief that cystic disease and solitary cysts of the liver are different manifestations of the same disease. At any rate, it is generally accepted that the solitary cysts in nonarteriosclerotic kidneys are but another expression of polycystic disease; a corresponding similarity would appear to extend to the manifestations of the disease in the liver. In several cases reported as solitary cysts of the liver, there have been small cysts in the proximity of the large cyst.

Nonparasitic solitary cysts of the liver have been observed at all ages, the majority of the cases occurring in adults, the oldest being that of Bland-Sutton,[6] who reported such an occurrence in a woman 75 years

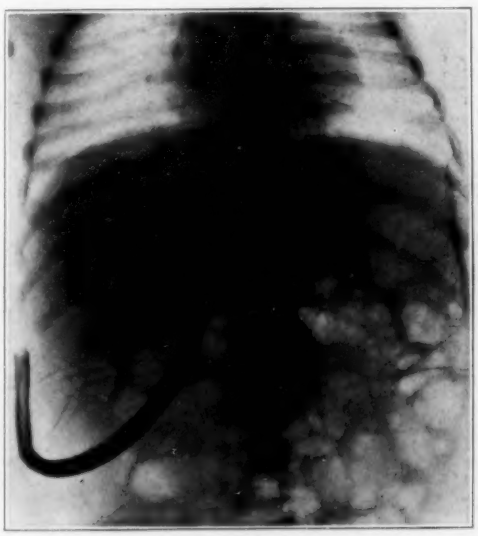

Fig. 8 (case 1).—Roentgenogram taken eleven days postoperative, indicating that little opaque solution could be injected through tube in wound.

of age. In reviewing the reported cases of solitary cysts in which operation has been performed, we find that three occurred during the first decade of life and six during the second. In 1903, Miller [10] observed a girl 2½ years old, with an enlarged abdomen dating from birth. Five pints (2.8 liters) of fluid, thought to be bile, was removed, and the patient recovered. In 1909, Shaw and Elting [9] reported a case of nonparasitic solitary cyst of the liver in a girl aged 1½ years. This patient had an abdominal swelling which was increasing in size, and by incision and drainage 900 cc. of clear, yellow fluid containing a mod-

10. Miller, G. B.: Congenital Dilatation of the Gall Bladder and Bile Ducts, Am. J. Obst. **48**:182 (Aug.) 1903.

erate amount of albumin, was removed. The child died from shock, and postmortem examination revealed normal kidneys. In 1918, Lowenburg [11] published an instance of a boy aged 19 months, who had a large firm mass occupying almost the entire abdomen. At operation, a large cyst containing bloody fluid was found.

The disease occurs more often in the female, roughly three times as many cases occurring in females as in males. According to Boyd,[1] this preponderance of the condition in the female sex is in agreement with the fact that developmental defects are considered to be more common in that sex. Prof. R. E. Scammon [12] stated that this is the case, apparently, for male infants with congenital malformations appear to die more often than females.

Fig. 9 (case 1).—Roentgenogram taken six weeks postoperatively, with inflation of colon outlining a larger liver shadow, indicating marked regeneration.

The cysts vary considerably in size; sometimes they assume enormous proportions. Witzel [13] reported an instance in which a large cystic liver prevented normal delivery of a child. The physician in attendance on the woman had pulled the head off the fetus in attempting to deliver it, and Witzel found it necessary to employ the perforator before he was able to extract the remainder of the fetus. The liver was

11. Lowenburg, H.: Congenital Unilocular Cyst of the Liver, Arch. Pediat. **35**:285 (May) 1918.

12. Scammon, R. E.: Personal communication.

13. Witzel, O.: Hemicephalus mit grossen Lebercysten, Cystennieren, und einer Reihe anderer Missbildungen, Centralbl. f. Gynäk. **4**:561, 1880.

cystic, and as large as the normal liver of an adult. The kidneys also were cystic, and many other abnormalities were present. Sänger and Klopp [14] and Bagot [15] each had a case of large cystic liver which was a hindrance to normal delivery of the child. Sänger and Klopp [14] stated that in their case the woman went into labor, but the child could not be born. Energetic traction was finally made, after it was found that the fetus was dead. Suddenly, the abdomen of the fetus ruptured and the uterine contents were expelled. Examination of the abdominal contents later revealed five cysts; two of these were definitely identified as cysts of the liver, one cyst being the size of a child's head. The other three

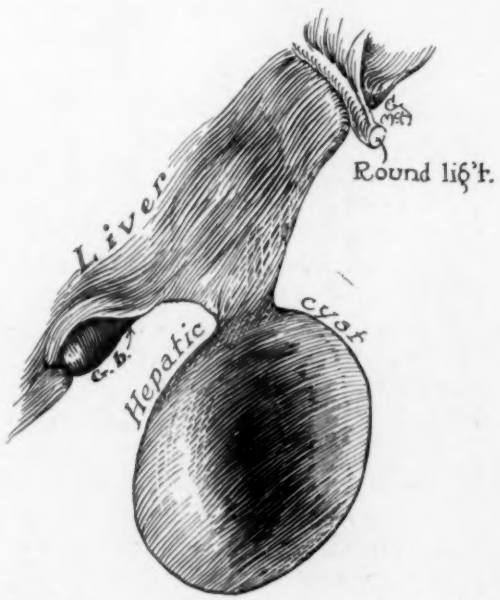

Fig. 10 (case 2).—Diagrammatic sketch of the relation of the hepatic cyst to the liver.

cysts were small and were more closely related to the intestine. In Bagot's [15] case, the delivery of the fetus was accomplished only after the abdomen of the infant was perforated and the fluid in the cyst allowed to escape. Cousins [16] reported an instance in which a cyst of the liver contained 2½ gallons (11.3 liters) of clear, limpid, yellow fluid, and in

14. Sänger, M., and Klopp, A.: Zur anatomischen Kenntniss der angeborenen Bauchcysten, Arch. f. Gynäk. **16**:415, 1880.

15. Bagot, W. S.: Dystocia Due to a Cyst in the Liver of a Foetus, Dublin J. M. Sc. **93**:265 (April) 1892.

16. Cousins, J. W.: Large Hepatic Cyst Simulating an Ovarian Tumor, Brit. M. J. **11**:700, 1874.

Kaltenbach's case, according to Boyd, 320 liters of a viscid, greenish-brown fluid was removed by successive punctures. In the instance reported by von Haberer,[17] a cyst occupied the whole left lobe of the liver, extending up to the falciform-ligament, while in Bayer's [18] patient, the whole liver was involved.

The cysts apparently occur with greater frequency on the inferior surface of the liver, and the lobe near the gallbladder would appear to be the site usually elected by the smaller cysts.

Examination of the nonparasitic solitary cysts shows the wall of the cyst to be thin, varying usually from 0.5 to 5 mm. in thickness. The external surface is usually smooth in contour, but bands of connective tissue have been observed to run over the cyst, producing areas of constriction, between which areas of definite bulging may occur. The cyst may be attached to adjacent organs by delicate bands of connective tissue, while it may be bound to the liver by a rather extensive amount of connective tissue. Good microscopic descriptions of the walls of nonparasitic cysts of the liver have been furnished by Plenk,[19] Sonntag [7] and Schaack.[20] Three layers of connective tissue can often be distinguished: (1) An inner layer of loose tissue, rich in cellular elements; (2) a circularly arranged, dense layer, poor in cell nuclei, and containing a few blood vessels with thickened intima; (3) a loose outer layer with elastic fibers, muscle bundles, rich in cells, many blood vessels and a few bile ducts. The cyst may be lined by a layer of cubical epithelium, although in some cysts this epithelial lining is missing. The bile ducts found in the wall of the cyst are lined usually with the same kind of epithelium which lines the cyst. That portion of the cyst which is in close relationship with the liver is found on microscopic examination to have the outer layer of connective tissue of its wall fusing with a portion of liver, which presents a picture closely resembling a mild cirrhosis. Nests of aberrant bile ducts and compressed liver cells are found close to the wall of the cyst.

The fluid obtained from the nonparasitic solitary cysts is clear, watery, sometimes yellowish-brown, containing albumin, mucin, cholesterol, blood, hematoidin, tyrosin, granular débris and occasionally, bile. In explanation of the presence of bile in such cysts, Boyd ventured to say that as the cyst increases in size, large intrahepatic bile ducts rupture into the cyst, their walls being destroyed by pressure. This

17. Von Haberer, H.: Zur Frage der nichtparasitären Leberzysten, Wien. klin. Wchnschr. **22**:1788, 1909.

18. Bayer, C.: Ueber eine durch Operation geheilte ungewöhnliche grosse Lebercyste, Prag. med. Wchnschr. **17**:637 (Dec.) 1892.

19. Plenk, L.: Zur Kenntnis der solitären Leberzysten, Virchows Arch. f. path. Anat. **201**:335, 1910.

20. Schaack, W.: Zur Frage der nicht parasitären Leberzysten, Arch. f. klin. Chir. **125**:183, 1923.

explanation would appear to find support in the fact that following drainage of the cyst, bile has been known to escape through the drainage tube, when the contents of the cyst at operation failed to show bile pigments.

A study of the literature shows that many consider the cysts to be adenomatous outgrowths of the bile ducts. Siegmund [21] was perhaps the first writer to present the idea that cysts were true adenocystomas. Von Kahlden [22] and Neuwerk and Hufschmid [23] upheld the neoplastic theory of cysts of the liver, basing their conclusions more on a study of cystic kidneys. All of these authors share the opinion that the cysts develop primarily as a result of some disturbance in the growth of the epithelium of the bile ducts of the liver. Borrmann,[24] however, insisted that both the epithelium and the connective tissue are concerned, there being a congenital disturbance in growth or development. Kaufmann,[25] Konjetzny,[26] Sternberg [27] and others designated the condition as "cystadenoma hepates." Plenk [19] claimed that the cysts do not arise from functioning ducts, but are derivatives of the excessive or aberrant bile ducts. Rolleston and Kantback [28] presented the theory that certain bile ducts are pinched off through an intra-uterine inflammatory process. Recently, Burns [29] has found that the liver tissue surrounding some of the cysts is infiltrated with many small round cells, indicating an early chronic inflammatory reaction.

Some authors have considered nonparasitic solitary cysts of the liver as retention cysts. Jones [2] quoted Virchow as believing that either calculi or cicatrices, by causing a narrowing of the bile ducts, permit the development of retention cysts. Jones [2] considered his case to be a retention cyst of the liver, although no sign of obstruction or narrowing

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21. Siegmund, A.: Ueber eine cystische Geschwulst der Leber, Virchows Arch. f. path. Anat. **115**:155, 1889.

22. Von Kahlden, C.: Ueber die Genese der multiloculären Cystenniere und der Cystenleber, Beitr. z. path. Anat. u. z. allg. Path. **13**: 291, 1893.

23. Neuwerk, C., and Hufschmid, K.: Ueber das multiloculäre Adenokystom der Niere, Beitr. z. path. Anat. u. z. allg. Path. **12**:1, 1893.

24. Borrmann, R.: Zur Frage der cystischen Entartung der Leber, Biblioth. Med. **13**:1, 1900.

25. Kaufmann, E.: Lehrbuch der speziellen pathologischen Anatomie, ed. 6, 1911, vol. 1, p. 608.

26. Konjetzny, G. E.: Pathologische Anatomie und Physiologie der Gallenblasen und Gallengangserkrankungen, Ergeb. allg. Path. u. path. Anat. **14**:712, 1910.

27. Sternberg, C.: Pathologische Anatomie, Ludwig Aschoff, ed. 5, 1921, vol. 1, p. 928.

28. Rolleston, H. D., and Kantback, A. A.: Cystische Erkrankung der Leber in Neugeborenen, Virchows Arch. f. path. Anat. **130**:488, 1892.

29. Burns, C. R.: Case of Multiple Cystic Liver, J. Path. & Bact. **28**:392 (April) 1925.

of the bile ducts was found. He stated that possibly an unrecognized irritation had caused swelling and retention in a bile duct, with the occlusion of its outlet. Recently, in 1925, Orr and Thurston [4] reported strangulation of a nonparasitic solitary cyst of the liver attached to the latter by a pedicle, and referred to the cyst as a retention cyst. Although we are not concerned primarily with the nature of the origin of these cysts, it would appear to us that the term "retention cyst" may be badly chosen; especially as the contents of the cyst are frequently neither bile nor mucus, the only two secretions retained behind an obstructed bile duct. If these cysts were true retention cysts, one should expect to observe bile or mucus escape through the drainage tube regularly following marsupalization, even though neither bile nor mucus were contained in the cyst at the time of operation. The small cysts occasionally seen on the external surface of the liver, beneath the capsule, in obstructive jaundice, may more properly be called true retention cysts.

SYMPTOMS AND DIAGNOSIS

Solitary nonparasitic cyst of the liver usually gives rise to symptoms only when, because of its size, pressure phenomena occur. Mild gastrointestinal disturbances, including nausea, occasionally vomiting, and diarrhea have been reported. The patient may complain of pain in the upper part of the abdomen.[30] Jaundice is rarely observed, and occurs only when the cyst is large enough to exert pressure on the extrahepatic bile ducts. A swelling usually most prominent in the right upper half of the abdomen may be seen on inspection, and a mass felt in this region on palpation. A cyst in the left lobe of the liver would be easily confused with a pancreatic cyst.

According to Jones,[2] Kilvington [31] of Melbourne (where hydatid cysts are common) made the correct preoperative diagnosis in an instance of nonparasitic cyst, his criterion being that the fluid tension was less than in the ordinary hydatid cyst. The hydatid thrill formerly believed to occur frequently in echinococcus cysts, is so rarely obtained by experienced observers in such cysts as to be of little diagnostic value (Huber, F. Deve and Barnett, quoted by Jones). Consequently, its absence is of no value in the differential diagnosis. Cystic dilatation of the common bile duct has to be thought of in the differential diagnosis. Jaundice, pain and fever are frequently present with such cysts, however, whereas these symptoms are rarely observed in nonparasitic solitary cysts of the liver. Ovarian cysts, cysts of the mesentery,

30. In a series of cases of multilocular cyst of the liver, reviewed by Caylor of the Mayo Clinic, pain was a common complaint. (S. Clin. N. Amer. **9**:191 [Feb.] 1929).

31. Kilvington, B.: An address, M. J. Australia **2**:555, (Dec. 17) 1921.

pancreatic cysts, cysts of the kidney, tumors of the liver, hydrops of the gallbladder and tuberculous peritonitis are found as the usual preoperative diagnoses in the reported cases. Hofmann [32] has pointed out that in large tumors of the liver, the colon is pushed down and to the left, while in the case of a large tumor of the kidney, the colon lies over the swelling. This point we believe to be of immense value in determining whether the tumor is in the kidney or liver, and by inflating the colon with air, the relation of the colon to the tumor may easily be made out. Movement of the cyst with respiration helps to decide whether the position of the tumor is influenced appreciably by the excursions of the diaphragm, in which event it would be likely to be found within the peritoneal cavity. In the instance of the large cyst reported by us, however, the cyst filled the greater portion of the abdomen, pushing the diaphragm up and practically inhibiting its excursion.

TREATMENT

The only treatment for solitary cysts of the liver demanding relief is surgical intervention. Excision of the cyst should be done when the nature of its attachment to the liver permits. Von Haberer [17] extirpated the entire left lobe of the liver in an instance in which such a cyst had practically replaced the liver tissue. Harrington enucleated successfully a multilocular hemorrhagic cyst of the liver without rupturing it. This cyst extended deep into the substance of the liver and involved the right lobe more than the left. When it was removed, a large cavity was left in the center of the liver. There was considerable oozing of blood, and the cavity was packed with gauze. Hofmann,[31] in removing a nonparasitic cyst of the liver, removed also a portion of the liver adjacent to the cyst, recovery occurring. Boyd [1] stated that Kaltenbach and König performed the same operation in their cases. Drainage with partial resection of the cyst is less dangerous and gives favorable results. This procedure also permits of removal of a piece of the wall of the cyst for microscopic examination. Faltin [5] modestly reported having unexpectedly found a large cyst of the liver, when operating for a supposed hydrops of the gallbladder, and being unfamiliar with the nature of the condition, closed the abdomen and later successfully removed the cyst.

Kilvington [31] stated that incision, removal of the fluid and daughter cysts and closure without drainage, after thorough swabbing of the lining of the wall of the cyst with a diluted solution of formaldehyde U. S. P. (1:10), is growing popular in Australia in the treatment

32. Hofmann, C.: Ueber wahre Cysten der Leber mit besonderer Berücksichtigung der klinisch bedeutungsvollen Cystadenome, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **10**:476, 1902.

of hydatid cysts. This plan, he reported, has shortened the convalescence following operation, and is equally as successful as external drainage. This method, so far as we know, has not been employed in the treatment of nonparasitic cysts.

In some instances in which external drainage has been done, a sinus has remained for from a few months to years. Jones [2] stated that in Theodoroff's case, a sinus obtained for twenty-seven months following drainage of a nonparasitic cyst. In Evans'[33] case, a sinus persisted. Porter's [34] patient developed a chronic sinus, and the patient died following an attempt to correct this surgically. In some instances, death from shock has occurred following operation. In Guerkens' case (quoted by Jones), the patient died after removal of such a cyst, and Shaw and Elting's [9] patient died of shock following incision and drainage.

We have had occasion to observe in a number of instances that the sudden opening of the abdomen and quick evisceration of a large cyst or tumor mass in children has resulted in shock. Scott [35] and one of us (O. H. W.) studied this phenomenon experimentally in animals. Following the establishment of an acute experimental ascites in dogs, it was noted that sudden decompression of the distended abdomen was attended by a marked fall in blood pressure. By decompressing the abdomen slowly, this fall in blood pressure was averted, and when pituitary extract was administered subcutaneously prior to the sudden release of intra-abdominal pressure, shock did not obtain.

Careful opening of the abdomen and deliberate evacuation or evisceration of a cyst or tumor mass is, therefore, more compatible with the patient's safety than a hurried entry into and exit from the abdomen by the surgeon.

Murchison (quoted by Jones) is said to have observed suppuration in a nonparasitic cyst of the liver that opened into the peritoneal cavity, causing death. When puncture and aspiration was the method by which patients with large intra-abdominal cysts were relieved of dyspnea and other pressure symptoms, peritonitis was an occasional sequel. Jones [2] stated that death resulted from this complication in two cases of nonparasitic solitary cysts of the liver (cases of Glotz and North) in which puncture and aspiration were employed.

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33. Evans, A.: Cystic Adenoma of the Bile Ducts, Brit. J. S. **9**:155, 1921.

34. Porter, C. B.: Adenocystoma of the Liver, Boston M. & S. J. **142**:427 (April 26) 1900.

35. Wangensteen, O. H., and Scott, H. G.: Collapse Following Sudden Decompression of the Distended Abdomen: A Study in Experimental Ascites, Arch. Surg. **16**:144 (Jan.) 1928.

SUMMARY

Two cases in which the patients were successfully operated on for solitary nonparasitic cysts of the liver are added in this report to those already on record. Solitary nonparasitic cysts of the liver are allied to cystic disease, but the nature of their origin remains obscure. These cysts occur with predilection in the female, and rarely give rise to symptoms other than those of pressure. Only once has the correct preoperative diagnosis been made. The attachment to the liver of large abdominal cysts may be recognized in that such swellings in the right lobe of the liver push the ascending colon, hepatic flexure and the transverse colon downward and to the left. The treatment of election, when the nature of the attachment of the cyst to the liver permits, is removal, but excision of a part of the wall of the cyst, marsupalization and drainage is a satisfactory mode of treatment with which the surgeon has more often to content himself.

SUCCESSIVE ROENTGENOGRAMS OF THE CHEST OF CHILDREN DURING MEASLES *

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The frequency of symptoms in measles that are referable to the respiratory tract is generally recognized. Little or nothing is known of the roentgenologic changes that occur in the chest during the various stages of the disease. Our purpose in this study, therefore, was to determine by roentgenologic methods the frequency of changes in the lung, the character of these changes and, finally, their progress.

The study is based on the examination of 130 children who were admitted to the Willard Parker Hospital because of measles. Only children admitted during the preeruptive or eruptive stage are included in this series. In a subsequent study, we shall report further data on the roentgenograms of these patients, made from four to eight months after their discharge from the hospital.

METHOD

Successive roentgenograms of the chest were taken every two or three days during the child's stay in the hospital. In all, 388 roentgenograms were studied. In thirteen cases, the first roentgenograms were taken during the preeruptive stage; in forty-six, during the early eruptive stage; in fifty-six, during the height of the eruption, and in fifteen, during the recession of the eruption. A physical examination was made by one of us whenever a roentgenogram was taken. The ages of these children ranged from 7 months to 12 years; eighty-five were less than 4 years of age, and forty-five were from 4 to 12 years of age.

The roentgen technic was as follows: The exposures were made at a distance of 115 cm. (46 inches), using a 15 cm. (6 inch) point spark gap with 40 ma. of current. The time of exposure was one tenth of a second. Except in a few instances, the roentgenograms were taken in the posterior-anterior position.

ROENTGENOLOGIC OBSERVATIONS

The following features were noted in the roentgenograms: (1) shadows in the lung suggesting infiltration; (2) abnormal intensity of the pulmonary markings; (3) pleuropulmonary changes; (4) progressive and retrogressive changes of the hilar area during the course of the disease, and (5) no deviation from normal.

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* From the Willard Parker Hospital for Contagious Diseases.

* Read before the Pediatric Section, New York Academy of Medicine, May 9, 1929.

Pneumonic Infiltrations.—Shadows suggesting infiltration of the lung were seen at some time during the preeruptive or eruptive stage of measles in sixty-four cases, and soon after the eruption had disappeared in eight cases. In the remaining fifty-eight cases, no pneumonic shadows were present. Pneumonic infiltrations were observed more frequently in younger children. Fifty-three (62.2 per cent) of the eighty-five children less than 4 years of age had pneumonic infiltration, whereas only nineteen (42.2 per cent) of the forty-five children who were 4 years of age or older had infiltrations.

TABLE 1.—*The Relation of Roentgenographic Pulmonary Infiltration to the Age of the Patients*

Age in Years	Total Number of Cases	Number of Cases with Pneumonic Infiltration	Percentage with Pneumonic Infiltration
Under 1	11	8	72.7
1–2	32	22	68.8
2–3	19	11	57.9
3–4	23	12	52.2
4–5	13	4	30.8
5–6	11	4	36.4
6–7	10	5	50.0
7–12	11	6	54.5
Totals	130	72	55.4

TABLE 2.—*The Relation of Roentgenographic Pulmonary Infiltration to the Stages of Measles*

Stage of Measles	Total Number of Cases	Number of Cases with Pneumonic Infiltration	Percentage with Pneumonic Infiltration
Preeruptive	13	3	23.0
Early eruptive	59	11	18.7
Full eruptive	115	44	38.3
Receding eruptive	130	64	49.3
Posteruptive	130	72	55.4

In thirteen cases, roentgenograms were taken for the first time during the preeruptive stage; three already showed pulmonary infiltration. In forty-six cases, roentgenograms were taken for the first time during the early eruptive stage; eight already showed infiltration. In fifty-six cases, roentgenograms were taken for the first time during the height of the eruption; nineteen showed pulmonary infiltration. In fifteen cases, roentgenograms were taken for the first time during the receding stage of the eruption; four showed pulmonary infiltration (table 2 and fig. 1). It is evident, therefore, that pneumonic infiltrations are frequently seen early in the disease; in some cases, it is seen even in the preeruptive stage. In fact, in forty-four of the seventy-two cases (61 per cent) in which infiltration was observed, it was already present before or during the height of the eruption. In sixty-six cases, no infiltration was seen during the eruptive stage. On further roentgen examination, however, eight

of these sixty-six cases showed an infiltration after the eruption had disappeared. Thus it is seen that pulmonary involvement is characteristic of the preeruptive and eruptive stages, and only exceptionally are pulmonary infiltrations found for the first time during the posteruptive period.

The shadows in the lung suggesting pulmonary infiltration were of various types: (1) disseminated confluent fine and coarse mottling in various areas of the lung fields, (2) large homogeneous triangles and (3) large homogeneous opacities obscuring the hilar and paravertebral regions.

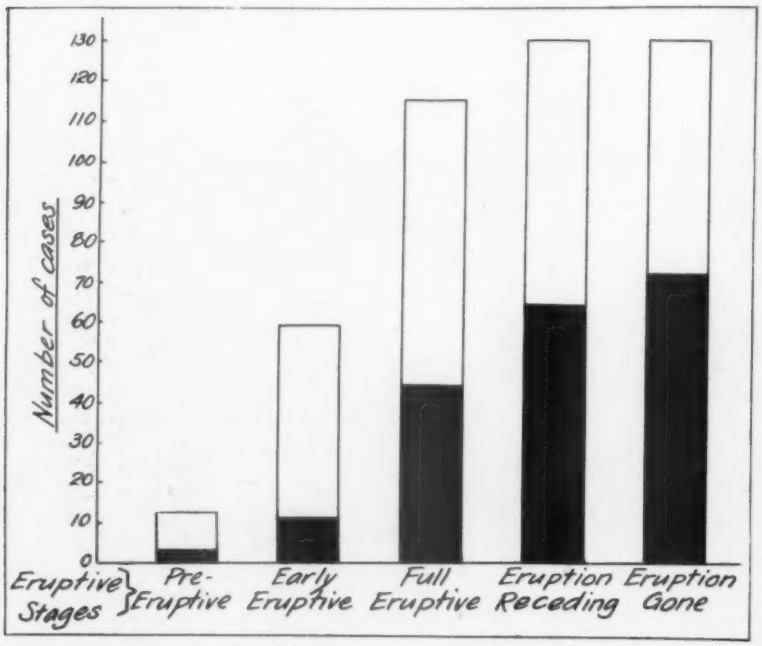

Fig. 1.—Graphic representation of the number of cases showing pneumonic infiltration by eruptive stages. The entire bar shows the total number of cases; the black portion, the cases showing pneumonic infiltration.

Disseminated or single patches of infiltration visualized in various areas of both lung fields, as usually seen in lobular pneumonia, occurred in sixty-eight cases, sixty-three times as the only type and five times with another type of infiltration. Successive roentgenograms seemed to indicate that these disseminated areas of fine and coarse mottling increased in extent and density coalescing to form confluent patches of pneumonic infiltration (figs. 2, 3 and 4). The cardiophrenic angles were common sites of infiltrations. The roentgenographic appearance of the shadow at the cardiophrenic angle was as follows: A diffuse

irregularly shaped opacity was seen over the base of the lung, close to or confluent with either of the cardiophrenic angles and partly obscuring the diaphragm but never spreading to the costophrenic sinus. These opacities were independent of the coarse linear strands extending downward from the lower pole of the hilum to the diaphragm (fig. 5). This type of lesion was observed in twenty-seven cases. The cardiophrenic shadows were observed as the only infiltration in twenty cases. It was more frequent on the right side than on the left. In fourteen cases, it was observed on both sides. One must remember that the left lower lobe of the lung, especially in children, is to a considerable extent overshadowed by the heart. A basal infiltration in this region can exist and not be visualized roentgenographically, when the roentgenogram is taken in the anterior-posterior position. This may account for the apparent greater frequency of this shadow on the right side.

Fig. 2.—*A* shows the lungs of J. C., aged 3 years, on June 8, during the preeruptive stage. The temperature was 104 F. There were no râles in the chest. The roentgenogram shows slight enlargement of both hilar regions. *B* was taken on June 11, during the early eruptive stage. The temperature was 103 F. There were coarse râles throughout the chest. The roentgenogram shows an increase in the size and density of the hilar shadow and scattered areas of fine and coarse mottling throughout the mesial and inner thirds of both lung fields.

The large homogeneous triangular infiltrations were found in only six cases. The base of the triangle is usually at the axilla; the apex points toward the hilum (figs. 6 and 7). It was never found in children more than 3 years of age. It was noted twice as the only infiltration, and four times in association with some other type of infiltration. It occurred once during the early eruption, four times during the full eruption and once during the receding eruption. This shadow appeared four times in the right upper lobe and twice in the right lower lobe.

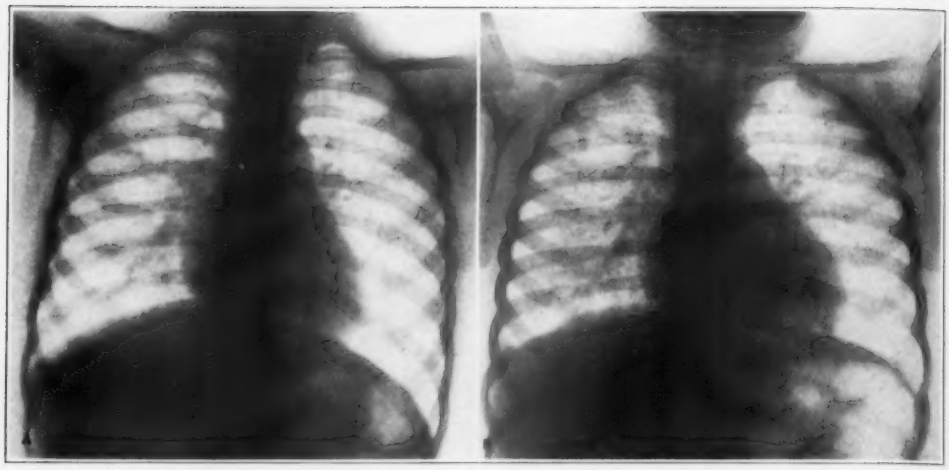

Fig. 3.—*A* shows the lungs of J. C., on June 13, at the height of the eruption. The temperature was 102 F. There were fine râles at the bases of both lungs posteriorly. The roentgenogram shows a further increase in size and density in the hilar shadow and an extension of fine and coarse mottling into the outer third of the lung field. *B* was taken on June 15, during the recession of the eruption. The temperature was 102 F. There were fine râles over the bases of both lungs. The roentgenogram shows a further progression of mottling with confluent areas of pneumonic patches, especially over the base and upper lobe of the right lung.

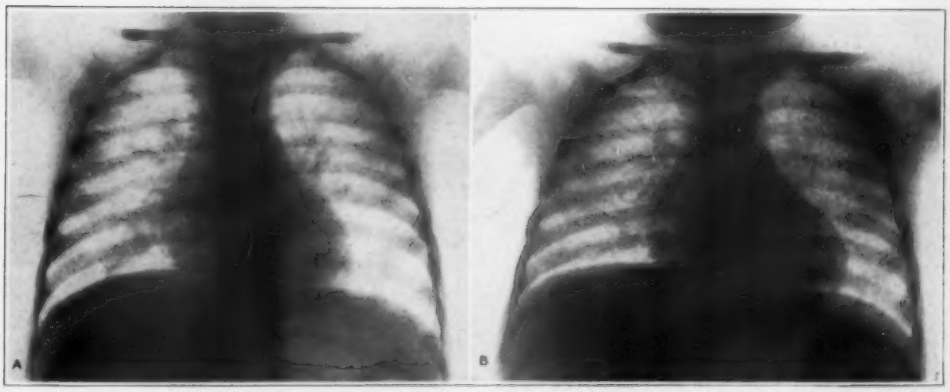

Fig. 4.—*A* shows the lungs of E. S., aged 15 months, on April 30, at the height of the eruption. The temperature was 99 F. On examination, the chest was normal. The roentgenogram shows scattered confluent coarse and fine mottling throughout the entire right lung and also the left upper lobe. A roentgenogram taken on April 28, when the patient's temperature was 101 F., shows about the same markings. *B* was taken on May 4 when the eruption was gone. The temperature was 99 F., and the chest was normal. The roentgenogram shows diminished illumination throughout the greater part of both lung fields with confluent patches in both upper lobes.

The large homogeneous opacities obscuring the hilar and paravertebral regions were observed in three cases, in two of which the patients were 1 year of age and in one, between 1 and 2 years. This shadow was seen twice as the only infiltration and once in association with a disseminated infiltration.

The most frequent site of infiltration in all the cases was the right lower lobe (forty-seven times). The right upper lobe (twenty-nine times), the left lower (twenty-five times), the left upper (twenty-three

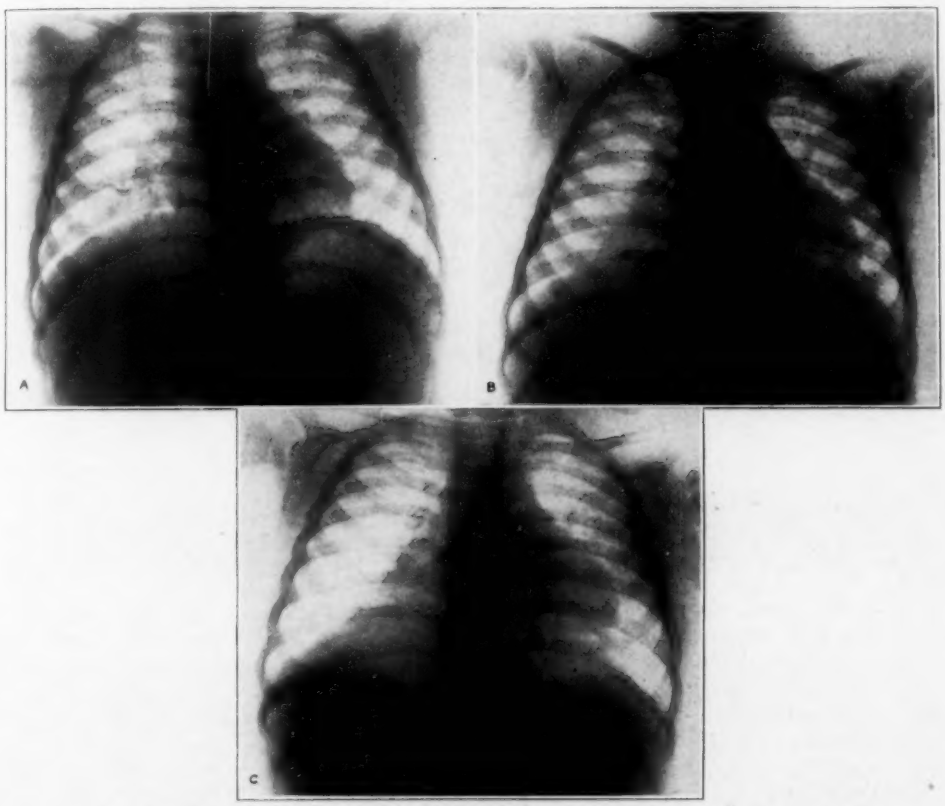

Fig. 5.—*A* shows the lungs of L. D., aged 18 months, on May 9, at the height of the eruption. The temperature was 104 F. There were fine râles at the bases of both lungs posteriorly. The roentgenogram shows a slight increase in the size and density of the hilar shadow. *B* was taken on May 11, when the eruption was receding. The temperature was 99 F. There were coarse râles at the bases of both lungs. The roentgenogram shows an area of opacity obscuring the right cardiophrenic angle and confluent with the hilar shadow. *C* was taken on May 14, when the rash was gone. The temperature was 99 F., and the chest was normal. The roentgenogram shows an extension of shadow over the base of the right lung.

times) and the right middle lobe (thirteen times) followed in order of frequency. In thirty-two cases, only one lobe of the lung was involved.

Progressive and retrogressive changes in the pulmonary shadows were observed in fifty-five cases during the course of disease. In seventeen cases there was no noticeable change in the roentgenographic appearance on subsequent exposures. The density of the infiltration was most intense in four cases during the preeruptive or early eruptive stage. In seventeen cases it was most intense during the height of the eruption, in twenty-three cases during the receding eruption and in eleven cases after the eruption was gone. The density of the shadow became most intense, therefore, during the receding and posteruptive stages.

In twenty-seven cases, shadows suggesting pneumonic infiltration first appeared and continued to progress after the temperature of the

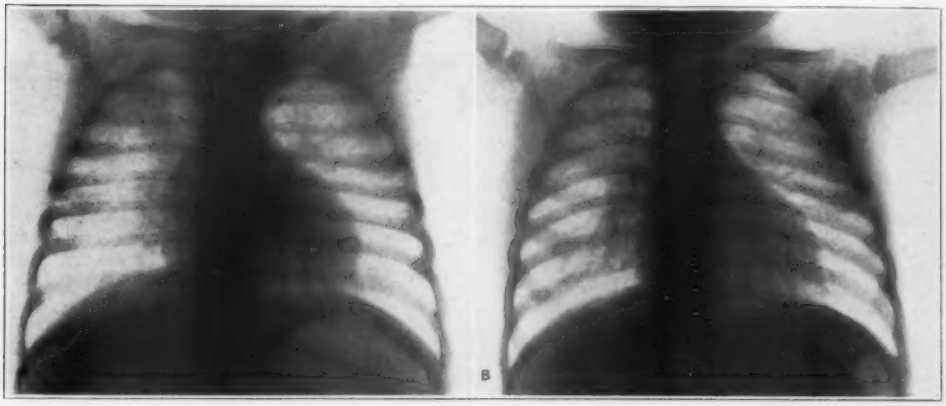

Fig. 6.—*A* shows the lungs of M. K., aged 14 months, on May 9, at the height of the eruption. The temperature was 104 F. There were coarse râles throughout the chest. The roentgenogram shows enlargement of both hilar regions, and an area of diminished illumination near the apex of the right lung. The base of the shadow is at the axilla and the apex toward the hilum. *B* was taken on May 11, when the eruption was receding. The temperature was 101 F. There were coarse râles throughout the chest. The roentgenogram shows an extension of the confluent infiltrative process of the right upper lobe.

child had returned to normal (fig. 5). Clinically these patients would have been regarded as having recovered from measles. In twelve of these cases, the results of physical examination of the lungs were completely negative. The others showed the bronchitic signs characteristic of measles.

It is interesting to attempt to correlate the presence of pulmonary infiltrations with the severity of the disease. In general, a greater percentage of infiltrations was found in severely sick children than in those with a clinically mild attack of measles. Thus, in the group in which

pneumonic infiltrations were absent, 79 per cent were mild cases, whereas only 44 per cent of the cases showing infiltration were considered as mild. Again, in the group showing infiltration the disease terminated fatally in seven cases. On the other hand, in the group showing no infiltrations there were no fatalities.

In similar fashion, we have tried to correlate the clinical observations on the chest with the occurrence of pulmonary infiltration. In the fifty-eight children in whom no pneumonic infiltration was seen in the roentgenograms, on physical examinations 14 per cent showed pulmonary or bronchial involvement. The most frequent signs, as so often found in measles, were scattered, coarse or fine, moist or dry râles. On the other hand, in the group of children in whom pneumonic infiltra-

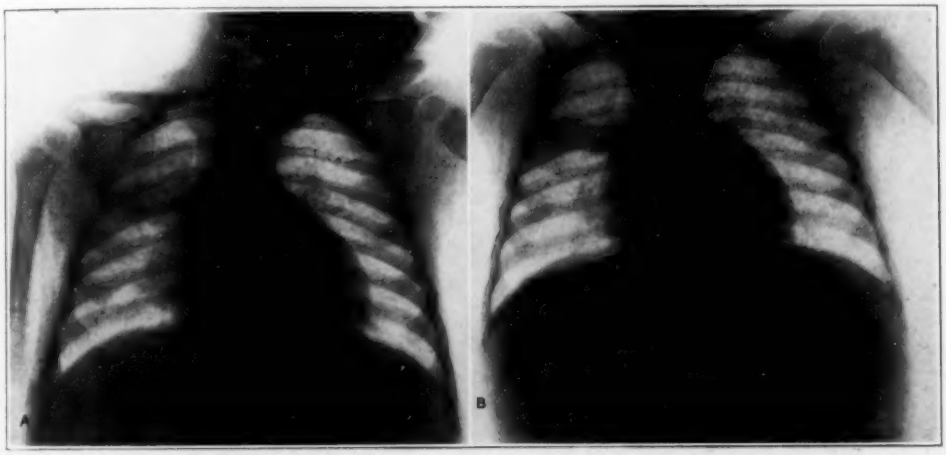

Fig. 7.—*A* shows the lungs of M. K., on May 14. The temperature was 99 F. There were fine râles anteriorly at the apex of the right lung. The roentgenogram shows increased density and size of the hilar shadow; also increased density of infiltration of the right upper lobe and scattered fine mottling around the infiltrated area. *B* was taken on May 16 when the eruption was gone. The temperature was 99 F. The roentgenogram shows the shadow of the right upper lobe to be denser.

tion was present according to the roentgenograms, a far greater number, 47 per cent, showed signs of bronchial or pulmonary involvement. However, in any given case, one could not predict from the physical examination the characteristics of the roentgenograms. Thus a negative clinical examination of the chest might be associated with a positive roentgenogram, and the presence of râles did not necessarily indicate the presence of roentgenologic pulmonary infiltration.

It was impossible to follow many of our cases for a sufficient length of time to determine the duration of the pulmonic infiltrations. However, in five cases in which the children were forced to remain in the

hospital because of some nonpulmonary complication, the shadows persisted for from two to four weeks.

Abnormal Intensity of Pulmonary Markings.—Abnormal intensity of the pulmonary markings was present in 80 per cent of all the cases. The roentgenographic appearance in children more than 4 years of age is somewhat different from that observed in children less than 4 years.

In the older children these markings assumed the appearance of coarse and fine linear striations radiating from the hilar regions into the pulmonary fields but not traceable to the extreme periphery (fig. 8). They varied in intensity, appeared early in the disease and, except in a few instances, were most intense at the height of the eruption or during its recession. In forty-three cases, progressive or retrogressive changes

Fig. 8.—The lungs of R. S., aged 6 years, on May 13, at the height of the eruption. The temperature was 104 F. There were fine râles low down in the right axilla and diminished breathing at the base of the right lung. The roentgenogram shows patchy, confluent mottling throughout both hilar regions and coarse beaded dense strands radiating toward the periphery but not reaching it.

in the intensity of the markings were noticed on the roentgenograms. In one case the markings were most intense during the preeruptive stage; in three, during the early eruption; in twenty-three, during the height of the eruption, and in sixteen, during the recession.

In younger children, the linear striations were not so definite. They appeared as fine beading following, presumably, the course of the pulmonary vessels (fig. 3 *A*). This beading or fine mottling was not confluent and it never reached the density of infiltrative opacities. As in the linear pulmonary markings of the older children, it was never traceable

to the extreme periphery. The striations appeared as early as the preeruptive stage, but they usually reached their greatest intensity during the height of the eruption.

Pleural Changes.—Interlobar shadows were observed in thirty-four cases (26 per cent). In twenty of these, they were associated with pulmonary infiltration. Of necessity these shadows were confined to the

Fig. 9.—Drawing of the lung of a patient with measles, who died three hours after the appearance of the eruption; × 6. There is peribronchial infiltration, but no infiltration around the blood vessels. *A* represents bronchioles. (From Dr. Denton's case.)

fissure between the right upper and middle lobes because visualization of the other fissures demanded a special technic not followed in this study. The interlobar shadows appeared only in the receding and posteruptive stages, never in the preeruptive stage.

Hilar Changes.—Because of the controversy concerning the significance of hilar changes, we shall merely report our results without ascribing to them any special significance. In thirty-five cases, the hilar shadow showed progressive and retrogressive changes while under observation. The hilar shadows were most intense, in the majority of the cases, during the height of the eruption. In ninety-six cases, a large homogeneous patchy density of the hilum was seen.

Involvement of the hilar lymph nodes was seen infrequently. It was never observed in children less than 4 years of age. Often these glands would become more clearly defined with the diminution in size and density of the hilar shadows.

COMMENT

It would seem, judging from the infiltrations seen on the roentgenograms of the chest, that pulmonary involvement during the preeruptive or eruptive stage of measles is frequent. What the exact nature of this process is, we do not know. The attempt to read from roentgenograms the exact nature of the pathologic process, oversteps the boundaries of roentgenologic interpretation. Nevertheless, the ultimate object is to link a particular roentgenologic appearance with clinical and pathologic changes. The roentgenogram does not necessarily reflect the actual pathologic lesion. Pulmonary shadows cast on the roentgenograms depend on numerous factors, such as the place and position of the part exposed, the summation of innumerable structures in one plane, and the intermingling of overaerated and underaerated areas. Interpretation, therefore, should be made with the utmost reserve.

Necropsies in the early stage of measles are few. Denton [1] had the rare opportunity of studying the pathologic process in a number of early fatal cases of measles during a severe epidemic in the Canal Zone. His observations correspond closely with those of MacCallum,[2] Koester,[3] Feyrter [4] and others. One of Denton's patients died three hours after the appearance of the rash; Koplik spots were present. The anatomic diagnosis was catarrhal tracheitis, capillary bronchitis, and cervical and mediastinal lymphadenitis. In the other two patients dying eighteen and twenty-four hours, respectively, after the appearance of the rash, early bronchopneumonia was found. Thus there is pathologic evidence of the occurrence of true bronchopneumonia during the early eruptive stage of measles. Unfortunately, no roentgenograms of the chest were

1. Denton, J.: Pathology of Fatal Measles, Am. J. M. Sc. **169**:531 (April) 1925.

2. MacCallum, W. G.: Pathology of Pneumonia in the U. S. Army Camps during Winter of 1917-1918, Rockefeller Institute, Monograph no. 10.

3. Koester: Masern pneumonie, Deutsche med. Wchnschr. **24**:8 V, 1898.

4. Feyrter, F.: Ueber die Masern pneumonie, Virchows Arch. f. path. Anat. **255**:753 (Aug.) 1924.

made. The gross appearance of the lungs of the child dying three hours after the appearance of the eruption, showed innumerable tiny red points varying in size from a fraction of a millimeter to 1 mm. These spots were so small that their relationship to sizable vessels and bronchial radicles could not be determined with the naked eye (fig. 9). Microscopically, there was seen in this early case an interstitial inflammatory process which started within the fibrous coats of the bronchioles. The inflammatory process spread through the peribronchial tissue into the adjacent alveolar parenchyma. Of course, in the later stages of measles, bronchopneumonia was seen.

The occurrence of pulmonary infiltrations during the preeruptive and early eruptive stages indicates the intimate association of infiltrations with the measles process, rather than with a secondary infection. This is confirmed by another observation in the present study. It will be recalled that infiltrations occurred for the first time after the temperature had returned to normal and when there was no evidence of any secondary infection. These data seem to support the theory of Denton,[1] Koester,[3] Feyrter [4] and others that in the early stages the pneumonic infiltration is a specific reaction of measles.

Single negative roentgenograms should not be interpreted as excluding the pulmonary infiltrations at some time during the course of measles. Our series includes negative roentgenograms in the early stages of the disease in cases in which positive observations are made later in the eruptive or posteruptive stages. For this reason, successive roentgenograms are essential in the study of the pulmonary changes in measles.

SUMMARY

1. Successive roentgenograms of the chest were taken of 130 children between the ages of 7 months and 12 years in the various stages of measles.

2. Pneumonic infiltrations were seen for the first time in the roentgenograms in the preeruptive, eruptive or posteruptive stages.

3. Pneumonic infiltrations were present in 62.4 per cent of the patients less than 4 years of age and in 42.2 per cent of the patients 4 years of age and over.

4. In the cases showing pulmonary infiltration, the infiltration was present in at least 61 per cent of the cases before or during the height of the eruption.

5. The pulmonary infiltrations were of three types: (*a*) large homogeneous triangles; (*b*) large homogeneous opacities obscuring the hilar and paravertebral regions, and (*c*) disseminated, confluent fine and coarse mottling in various areas of the lung field.

6. A common site for the disseminated type of infiltration was the cardiophrenic angles.

7. The most frequent site of infiltration was the right lower lobe; the right upper, the left lower, the right upper and the right middle lobes followed in the order given.

8. The infiltration was most dense, in the majority of cases, during the receding eruptive or posteruptive stage.

9. Shadows suggesting pulmonary infiltration first appeared and continued to progress in twenty-seven cases, even after the temperature of the child was normal. In none of these cases were there any definite physical pulmonary signs.

10. In the group in which no pneumonic infiltrations were observed, 79.2 per cent of the patients were considered as having had a clinically mild course of measles. Of the cases in which pulmonary infiltrations appeared in the roentgenograms, 44.4 per cent were considered clinically mild.

11. Physical signs giving evidence of bronchial or pulmonary involvement were present in 14 per cent of the group which showed no pulmonary infiltration on the roentgenogram and in 47 per cent of the pneumonic group. One could not predict from the physical examination during the eruptive stage the probability of finding a pneumonic infiltration in the roentgenogram.

12. These pneumonic shadows persisted for from two to four weeks after their appearance in five cases observed.

13. Abnormal intensity of the pulmonary markings was present in 80 per cent of all cases. Their roentgenographic appearance in children of 4 years and over was somewhat different from that observed in children less than 4 years of age. These markings were usually most intense during the height of the eruption.

14. In 26.2 per cent of all cases, there was an interlobar shadow between the right upper and lower lobes of the lung. The shadow was seen only during the latter stages of the disease.

15. In 74 per cent of all the cases, there was a large and dense hilar shadow. In thirty-five cases, the hilar shadow was seen to progress and retrogress roentgenographically while under observation.

16. In 20 per cent of the cases, no deviation from the normal was recorded on the roentgenogram.

17. Successive roentgenograms of the chest during measles are of great value.

55 East 86th Street.

THE POLYMORPHONUCLEAR COUNT IN THE NEW-BORN

I. PRELIMINARY REPORT *

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Arneth [1] first pointed out that there were significant variations in the nuclei of the polymorphonuclear leukocytes which indicated with some precision the severity of the toxemia in an infectious disease. Unfortunately, his method of classification was so complicated that it afforded no sure basis for clinical work. Cooke [2] simplified the classification of the polymorphonuclear leukocytes so that the method can be carried out with comparative ease. In many studies carried out on the blood of adults, it was found that the count in the normal person falls within certain definite limits, and that in persons suffering from infectious conditions it shows characteristic changes. The blood of the new-born infant shows many differences from that of the adult, particularly in the cellular elements. It was therefore considered desirable to classify the types of polymorphonuclear leukocytes in the new-born.

METHOD

This study is based on a series of sixty new-born infants, and was carried on during a period of ten months. The differential counts were made within six hours after birth and at twenty-four hour intervals thereafter. The determinations were made at the same time every day. The slides were stained with carbol-pyronin,[3] the films being dried in air and the stain applied for fifteen minutes. No preliminary fixation was required after washing and drying. They were examined unmounted. The stain imparted a deep blue color to the nuclei and a pink color to the cytoplasm. The red cells stained feebly pink.

In drawing the blood for making determinations the skin of the heel was thoroughly cleansed with green soap and water, and finally with alcohol, and a deep puncture was then made in the heel, which resulted in an adequate flow of blood. To insure a free flow, 3 drops were allowed to fall as a routine.

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* Submitted for publication, March 2, 1929.

* From the Department of Pediatrics, Rush Medical College of the University of Chicago, and the Presbyterian Hospital.

1. Arneth, J.: Technik des Blautausstriches und eine neue Differentialzahltrafe für Leukozyten, Deutsche med. Wchnschr. **29**:2560, 1913.

2. Cooke, W. E.: The Nucleus of the Neutrophil Polymorphonuclear Leukocyte in Health and Disease, J. Path. & Bact. **29**:494, 1914-1915.

3. Cooke, W. E., and Ponder, Eric: The Polynuclear Count, Philadelphia, J. B. Lippincott Company, 1927.

Cooke's classification, which he has termed "the polynuclear count," was used. The polymorphonuclear leukocyte with an undivided nucleus was placed in class I; the cell the nucleus of which had divided into two lobes in class II; the cell with three lobes in class III, and the cell with four lobes in class IV. Nuclei without a cell body or those subjected to pressure were not counted. One hundred cells were counted in each slide. That the changes in the various classes might be appreciated better, the weighted mean devised by Cooke and Ponder was used. This was obtained by multiplying the number of cells in class I by one, those in class II by two, those in class III by three, and those in class IV by four. The results were added and divided by the total number of cells.

COMMENT

Cooke,[4] in a differential study of the polymorphonuclears, determined the normal count in the adult by taking the polymorphonuclear count in ninety normal persons. The average number of cells found was as follows class I, 12; class II, 25; class III, 44; class IV, 15 and class V, 4.

Results

Day	Distribution of Polymorphonuclear Leukocytes				Mean
	Class I	Class II	Class III	Class IV	
1	57	40	3		1.43
2	61	37	2		1.41
3	50	43	7		1.57
4	50	42	8		1.58
5	56	36	8		1.52
6	55	35	10		1.55
7	50	32	18		1.68
8	47	37	16		1.69
9	42	28	25	5	1.93
10	33	36	22	9	2.07

The average weighted mean was 2.74. The polymorphonuclear population of the blood vessels is only a fraction of the whole population of circulating polymorphonuclears in the body. These cells abound in the extravascular spaces, from which they are continually passing from the vessels. The cells of class I, and occasionally those of class II, are the only ones that are formed in the hemopoietic system. These later mature in the blood stream, becoming the older cells of classes II, III and IV. Therefore, the cells of classes I and II are definitely the young cells, and in the adult show the first results of any change in the normal system. Cooke and Ponder stated that roughly in the normal adult the sum of the counts of cells of class I and class II should not exceed 40.

Arneth [5] made differential leukocyte counts on several infants over 9 days old, and found an absence of multinucleated leukocytes, and a

4. Cooke, W. E.: The Life History of the Neutrophile Polymorphonuclear Leukocyte, J. Roy. Micr. Soc. **47**:29 (March) 1927.

5. Arneth, J.: Qualitative Blutlehre, Arch. f. Gynak. **24**:1, 1920.

strong left-handed shift. This confirmed the earlier work of Esser,[6] who found the cells in an infant a few hours old to be 60 per cent of class I, 26 per cent of class II and 12 per cent of class III. Breinl and Priestley [7] had also found a strong left-sided shift in counts made on 150 children in Queensland. They attributed this to the warm climate, as there was a considerable leukopenia. Frank,[8] in an attempt to find some basic differences between breast-fed and artificially fed children, made leukocyte counts on a number from 8 to 10 days of age and found the left-handed shift to be marked. Arneth [9] again had the same results in some work done two years later. Fabris,[10] in work on thirteen infants from 1 to 5 days old, obtained results quite different. His average was: class I, 3; class II, 17; class III, 54; class IV, 21 and class V, 5 per cent. There is no explanation for this difference, but it will be observed that these percentages are far from those of even the normal adult.

In this series in the new-born, the polymorphonuclear counts showed a considerable variation from those in adults. On the first day, the cells of class I predominated and the sum of the numbers of class I and of class II was 97. The number of cells of class I fell rapidly to 33 by the tenth day, and the number of those of class II fell but slowly, so that they were more numerous than those of class I. The total number of the cells of class I and class II was then 69. The number of the cells of class III increased rapidly from the first day, and cells of class IV appeared on the ninth day. As no increase was seen in the number of cells of class I, but rather a sharp decrease, and as the total leukocyte count was not increasing but decreasing,[11] the cells of class III and of class IV, and in part of class II, must be derived from class I. In other words, the polymorphonuclear cells of the new-born are young cells that mature as the age of the child increases. It is shown that during the first ten days the cells mature from the single and double nuclei of the first two classes to the multilobed nuclei of the higher classes, or the older cells. Only three cells of class III are found on the first day, and none of class IV are found until the ninth day. This would indicate that at birth the new-born child's blood contains only those polymorpho-

6. Esser, M.: Das Neutrophile Blutbild beim natürlich und beim künstlich ernährten Säugling, München. med. Wchnschr. **34**:1651, 1906.

7. Breinl, A., and Priestley, H.: Note on the Arneth Count in Healthy Aboriginal Children, Ann. Trop. Med. **8**:565 (Oct. 1) 1917.

8. Frank, M.: Beitrag zur Biologie der Weissen Blut Zeller, in der Neubeburtszeit und in Saüglingsalter, Ztschr. f. Kinderh. **30**:16 (Nov.) 1921.

9. Arneth, J., and Neinkemper, D.: Ueber das normale qualitative Leukocytes Blutbild des Säuglings, Ztschr. f. Kinderh. **34**:263 (Jan.) 1923.

10. Fabris, S.: Il Quadro leukocitario qualitativo nel neonato secondo Arneth, Pediatria **31**:1025 (Oct. 1) 1923.

11. Sanford, H. N.: The Effect of the Ultraviolet Light on the Blood of the New-Born: III. White Cells, Am. J. Dis. Child. **37**: 1187 (June) 1929.

nuclear leukocytes that have been formed in the bone-marrow or other blood-forming organs of the hemopoietic system, and that the multilobed, or more mature, cells are not formed in the hemopoietic system but are the result of the younger, single lobed cells maturing in the blood stream.

The polymorphonuclear count of the new-born, however, undergoes rapid readjustment as the cells mature. This is best shown by the weighted mean. On the first day, when there is such a preponderance of young, single lobed cells of class I, the mean is only 1.43. This increases rapidly until the tenth day, when it is 2.07, indicating a marked tendency to arrive at the stable adult mean of 2.74.

RESULTS

1. The polymorphonuclear leukocytes of the new-born show a preponderance of the single and double lobed nucleated forms.

2. Only a few of the trilobed polymorphonuclear cells exist at birth, and none of the other multilobed forms appear until the ninth day.

3. There is a rapid tendency toward the adult mean.

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THE MANAGEMENT OF THE AMBULATORY DIABETIC CHILD*

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In the course of our experience in the care of ten ambulatory diabetic children over a period of two and a half years, we have been able to establish a procedure for the management of these patients of a fairly uniform nature. It was found expedient to prepare some simple tables from which the diets could be calculated and menus prepared. The methods employed in our course of treatment are based on the fundamental physiologic requirements of the growing child, the growth, activity and well-being of the patient being taken into account. The simplicity of the methods devised warrants recording them in the literature.

The importance of diabetes mellitus in children lies not so much in its frequency of occurence, for it is a relatively rare disease, as in its severity. A discussion of the etiology, pathology or diagnosis of this disease is beyond the scope of our paper. An excellent monograph on this subject has recently been presented by Priesel and Wagner[1] in which they reported their experience with 58 diabetic children. Joslin recorded his observations in a study of 100 children. Briefly, diabetes mellitus is a rare disease in children, occuring between the ages of 2 and 14. It is equally distributed between the sexes, has no particular racial affinity and is of unknown origin. It is characterized by a functional derangement in dextrose metabolism, attributed to disease of the pancreas. However, no structural changes have been observed in the pancreas which are compatible with the marked functional changes in metabolism that this disease discloses. We have tabulated the etiologic factors in our series of cases in table 1.

MANAGEMENT

After the diagnosis of diabetes mellitus has been confirmed, we prefer first to treat the patients in the hospital. There we establish the patient's tolerance by estimating the degree of glycosuria and acidosis, the blood sugar level and the response to a maintenance diet.

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* Submitted for publication, March 5, 1929.

* From the Pediatric Department and the Pediatric Research Laboratory, the Jewish Hospital of Brooklyn.

1. Priesel, R., and Wagner, R.: Ergebn. d. inn. Med. u. Kinderh. **30**:536, 1926.

TABLE 1.—*Etiologic Factors*

Name	Age at Onset in Years	Sex	Race	Family History	Time Between Onset of Symptoms and Discovery of Dextrose in Urine	Symptoms at Onset						Previous History
						Polyphagia	Polydypsia	Polyuria	Loss of Weight	Weakness	Other Symptoms	
W. S.	3	M	Scotch-Irish	Unimportant	3 months	++	++++	++++	±	±	None	Pertussis and otitis media
Y. K.	6½	F	Jewish	Unimportant	2 months	++++	++++	+++	++++	+	None	Measles and pertussis
J. J.	12	F	Jewish	Unimportant	5 years (?)	+	++	+++	0	0	Headache; drowsiness; vomiting; coma	Scarlet fever; diphtheria; measles; pertussis and chickenpox
I. C.	8	F	Jewish	Unimportant	3 weeks	++	++++	++++	+	++	Nausea; vomiting; dizziness; headache	Measles and pertussis
M. W.	8	M	Jewish	Paternal aunt	1 week	+	++++	++++	0	0	None	Measles; mumps; scarlet fever; occasional tonsillitis
A. E.	3½	M	Jewish	Unimportant	1 month	+	++++	++++	++++	++	Coma at onset	Bronchopneumonia; measles
S. C.	4½	M	Jewish	Maternal grandfather	3 months	+	++++	++++	++++	++	Drowsy and irritable	Measles; gastro-enteritis
Y. G.	10	F	Jewish	Paternal grandfather	1 month	+++	++++	++++	+++	+++	None	Measles; pertussis
H. P.	14	M	Jewish	Father mildly diabetic	1 week	++	++++	++++	+++	+	None	Chickenpox; measles; scarlet fever; pertussis; mumps
B. L.	11½	F	Jewish	Maternal grandmother had diabetes	6 months	++++	++++	+++	++++	+	Headache; pain in legs	German measles; measles; chickenpox; mumps; occasional sore throat

The patient is then transferred to the outpatient department, where management and progress are followed during weekly visits.

If the child is for any reason admitted simply as an ambulatory patient without hospitalization, the same laboratory investigation is conducted.

The aim in the treatment is to furnish the patient with a diet so constituted that he receives sufficient calories to increase his weight and to provide for physical activity as well as sufficient protein to provide for repair and growth of tissue and yet be within the limits of his own dextrose tolerance. If the child's tolerance is so low that he is incapable of utilizing the available dextrose in the maintenance diet, then insulin must be administered. There is also provision made in the diet to include the accessory food substances such as salts and vitamins.

Method.—The following data are first obtained: (*a*) height, (*b*) weight, (*c*) age, (*d*) sex, (*e*) degree or extent of glycosuria, (*f*) nature of the reducing substance excreted, (*g*) presence of ketone bodies in the urine, (*h*) fasting blood sugar and (*i*) the carbon dioxide combining power of the blood.

In addition to the foregoing data, the following items are stressed in the history and physical examination: hereditary factors, the presence of glandular disorders, anatomic anomalies, predominant symptoms, foci of infection and neurologic and vascular changes.

If the patient shows evidence of diabetes mellitus without the clinical signs of ketosis, he is given a test diet. The formula is so constructed that it complies with the following requirements: (1) a sufficient number of calories to provide for the total heat production of the body, (2) sufficient protein to provide for wear and tear of tissue, and for growth, (3) a minimum amount of available dextrose and (4) a ketogenic-antiketogenic ratio not greater than $\frac{1.5}{1}$

The caloric requirements are based on estimations of the basal heat production. In view of the fact that the basal metabolic rate in diabetes mellitus is normal, it is only necessary to utilize prediction tables which have been prepared. One can use the age-weight tables prepared by Benedict and Talbot [2] or the more recent tables of Holt and Fales.[3] We have found it practicable to use a table that we have compiled based on the figures of Lusk [4] and Holt and Fales (table 2). With patients over 12 years of age, the basal heat production is easily estimated from the Boothby nomographic chart [5] which is based on the surface area

2. Benedict, F. G., and Talbot, F. B.: Carnegie Inst. Washington Pub. no. 302, 1921, p. 206.

3. Holt, L. E., and Fales, H. L.: Food Requirements of Children, Am. J. Dis. Child. **22**:371 (Oct.) 1922.

4. Lusk, G.: J. Home Econ. **11**:281, 1919.

5. Boothby, W. M., and Sandeford, R. B.: Boston M. & S. J. **185**:337, 1922.

formula of Du Bois.[6] To the predicted basal heat production is added 10 per cent for the average specific dynamic action of the food consumed and from 50 to 100% for activity.

The protein intake depends on the age of the patient and is calculated as a percentage of the total calories (table 2).

From a table which we have prepared, the formula for the diet is read directly (table 3). This obviates the necessity of calculating the carbohydrate, protein and fat content of the diet. The amount of

TABLE 2.—*Caloric and Protein Requirements Based on Age and Activity and the Estimates of Graham Lusk, and Holt and Fales*

Age in Years	Basal	Very Quiet Child	Active Child	Average	Per Cent of Total Calories in Form of Protein
1	500	750	.....	950	15
2	800	1,200	1,600	1,400	15
3	900	1,300	1,750	1,500	15
4	950	1,400	1,850	1,600	15
5	1,000	1,500	1,900	1,700	15
6	1,100	1,650	2,200	1,800	15
7	1,200	1,750	2,300	1,900	15
8	1,250	1,800	2,400	2,000	15
9	1,300	1,850	2,500	2,100	10
10	1,350	2,000	2,650	2,300	10
11	1,400	2,050	2,750	2,500	10
12	1,450	2,150	2,850	2,600	10
13	1,500	2,200	2,900	2,700	10
14	1,500	2,250	3,000	2,800	10

TABLE 3.—*Method for Calculating Formula for Test Diabetic Diet* *

Protein..	5%			10%			15%			20%			25%			30%		
Calories	C. Gm.	P. Gm.	F. Gm.	C. Gm.	P. Gm.	F. Gm.	C. Gm.	P. Gm.	F. Gm.	C. Gm.	P. Gm.	F. Gm.	C. Gm.	P. Gm.	F. Gm.	C. Gm.	P. Gm.	F. Gm.
1,000	40	12	88	35	25	84	30	38	81	25	48	78	20	60	74	15	73	70
1,100	44	13	97	38	27	91	33	41	89	28	53	86	22	66	80	17	80	77
1,200	48	15	106	42	30	100	36	45	97	30	58	93	24	72	85	18	88	84
1,300	52	16	114	46	32	110	39	49	105	32	63	100	26	78	91	19	95	91
1,400	56	17	123	50	34	119	42	53	113	35	68	108	28	84	97	21	103	98
1,500	60	18	132	53	37	127	45	57	120	37	72	116	30	90	102	23	110	105
1,600	64	19	141	56	39	134	48	60	129	40	76	124	32	96	107	24	117	112
1,700	68	20	150	60	42	144	51	64	136	43	81	132	34	102	112	25	125	119
1,800	72	22	158	63	45	151	54	68	140	45	86	140	36	108	117	27	132	126
1,900	76	23	167	67	47	160	57	72	153	47	91	148	38	114	122	28	139	133
2,000	80	24	176	70	50	168	60	76	162	50	96	156	40	120	128	30	146	140

* This table can be utilized for caloric diets below 1,000 or above 2,000 calories by simple division or multiplication of the formula listed. Example: To calculate a diet of 800 calories the diet for 1,600 calories is divided by 2. Similarly to obtain the formula for a 2,400 caloric diet, read the diet for 1,200 calories and multiply by 2.

protein, carbohydrate and fat thus estimated will give the minimum amount of available dextrose, a ketogenic-antiketogenic ratio of $\frac{1.5}{1}$ and will comply with the necessary protein requirements.

The prescribing of a menu to contain the amount of food which would satisfy the formula evolved requires a knowledge of dietetics that the average physician usually does not possess. We have therefore prepared a table which is self explanatory and from which the menu can be read directly (table 4).

6. Du Bois, D., and Du Bois, E. F.: Clinical Calorimetry: V., Arch. Int. Med. **15**:868 (May) 1915; Clinical Calorimetry: X., ibid. **17**:863 (June) 1916.

TABLE 4.—*Estimated Amounts of Three Food Ingredients*

CARBOHYDRATE CHART

Carbohydrate, Gm.	5% Vegetables	10% Vegetables	10% Fruit	Uneeda Biscuits	Cereals	Bread	Potato or Banana	Milk	Protein Content of Diet*	Fat Content of Diet*
30	200	...	100	...	..	..	...	250	13	10
40	200	100	100	...	..	..	...	250	14	10
50	200	100	100	2	..	..	...	250	16	11
60	200	100	150	3	..	..	...	250	18	11
70	200	100	150	2	..	30	...	250	20	12
80	200	100	150	2	15	30	...	250	22	14
90	200	100	200	1	15	50	...	250	23	14
100	200	100	200	1	30	50	...	250	25	16
110	200	100	250	2	30	50	...	250	27	16
120	200	100	300	2	30	60	...	250	28	17
130	200	100	300	1	30	90	...	250	30	17
140	200	100	300	1	30	90	50	250	31	17
150	200	100	300	1	30	90	100	250	33	17
160	200	100	300	1	30	90	150	250	34	17
170	200	100	300	1	30	90	200	250	36	17
180	200	100	350	2	30	90	200	250	38	18
190	200	100	400	3	30	90	200	250	40	19
200	200	100	400	2	30	120	200	250	42	18

* Subtract protein and fat of Carbohydrate Chart from prescribed formula and the difference can be read from Protein and Fat Chart.

Directions: To give a diet containing 80 Gm. carbohydrate, 50 Gm. protein and 135 Gm. fat, read menu for 80 on Carbohydrate Chart. It will be seen that this menu contains 22 Gm. protein and 14 Gm. fat. Subtract 22 from 50, and 14 from 135, giving 28 protein and 121 fat, respectively. The closest figures approximating 28 protein and 121 fat are then read on the Protein and Fat Chart. Thus one selects the menu for 30 protein and 110 fat and adds 10 cc. of olive oil. This is accurate enough for clinical purposes.

PROTEIN AND FAT CHART

Protein, Gm.	Fat, Gm.	Cheese, Gm.	40% Cream, Cc.	Eggs	Meat or Fish, Gm.	Butter, Gm.	Olive Oil, Cc.	Lister's Bran Muffins†
20	30	10	...	2	30	15	..	..
	50	10	50	2	30	15	..	..
	70	10	100	2	30	15	..	..
	90	10	100	2	30	20	16	..
	110	10	125	1	30	30	20	..
30	40	15	30	2	50	10	..	..
	50	15	30	2	50	20	..	..
	70	15	75	2	50	20	..	..
	90	15	100	2	50	20	10	..
	110	15	100	2	50	30	20	..
	130	15	200	1	50	35	30	..
	150	15	225	1	50	45	30	..
40	70	15	100	2	75	20	..	..
	90	15	100	2	75	30	10	..
	110	15	150	1	75	30	10	..
	130	15	200	1	75	30	10	..
	150	15	200	1	75	40	20	..
50	90	30	100	2	100	20	..	..
	110	30	100	2	100	30	10	..
	130	30	100	2	100	40	20	..
	150	30	200	1	100	30	10	..
60	110	30	100	2	100	30	..	2
	130	30	100	2	100	50	..	2
	150	30	200	2	100	30	..	1

† Prepared so that one muffin is equivalent to one egg.

Note: To increase the fat content by 10 Gm. add 10 cc. of olive oil to the diet. To increase protein content by 6 Gm. add one egg. This, of course, also increases the fat content by 6 Gm.

Table 5 is a substitution table which contains a list of foods in portions that are equivalent to 100 Gm.

Example.—Table 2 shows that the total caloric requirements for a boy, aged 8 years, weighing 66 pounds (29.9 Kg.) and 50 inches in

TABLE 5.—*Substitution Table*

Vegetables—100 Gram Portions

3 to 5 per Cent		10 per Cent	
Asparagus, canned	5 tips	Beets	¾ cup
Asparagus, fresh	4 long stalks	Carrots	¾ cup
Beet greens, cooked	½ cup	Green peas, canned	½ cup
Celery	10 medium stalks	Olives	½ cup
Cucumber	10 medium slices	Onions	6 small
Endive	10 stalks	Oyster plant	¾ cup
Lettuce	10 large leaves	Pumpkins, canned	½ cup
Mushrooms	4 medium sized	Squash	½ cup
Romaine	10 large leaves	String beans, fresh	½ cup
Rhubarb, cooked	¾ cup	Turnips	¾ cup
Sauerkraut	¾ cup		
Spinach	½ cup		
Brussels sprouts	¾ cup		
Cabbage	1 cup		
Cauliflower	¾ cup		
Eggplant	1½ cup		
Green pepper	1 medium		
Radishes	5 small		
String beans, canned	½ cup		
Tomato, one	Size of egg		
Watercress	2 cups		

15 per Cent	
Artichokes	1 medium sized
Green peas, fresh	½ cup
Lima beans, canned	½ cup
Parsnips	¾ cup

Fresh Fruits—100 Gram Portions

10 per Cent		15 per Cent	
Orange	1 small	Apple	1 small sized
Grapefruit	½ small (4⅝ diam.)	Apricot	2 small
Cantaloupe	small dessert dish	Cherries	25 small
Peach	1 medium sized	Banana	⅔ of 1 medium sized
Pineapple	1 slice ¾ inch thick	Huckleberries	¾ cup
Strawberries	¾ cup	Blueberries	¾ cup
Watermelon	1 cup	Raspberries	½ cup
		Pear	1 small

Uncooked Cereals—10 per Cent Protein, 75 per Cent Carbohydrate

1. Barley (flour or whole grain)
2. Buckwheat (flour or whole grain)
3. Hominy
4. Rice
5. Wheat (farina, macaroni, noodles, Shredded Wheat, spaghetti)
6. Breakfast foods (Cream of Wheat, Grapenuts, Wheatena, Shredded Wheat Biscuit (1 biscuit weight 30 Gm.), hominy, Puffed Rice.)

Meat—20 per Cent Protein, 10 per Cent Fat

1. Chicken, broiler
2. Duck, breast
3. Pigeon meat
4. Round steak, fat removed
5. Veal (breast, leg, loin, etc.)
6. Leg of roast of lamb
7. Beef liver
8. Tongue

Meat—20 per Cent Protein, 20 per Cent Fat

1. Lamb (chops, roast)
2. Beef (boiled, corned, kidney, roast, steak, tongue)
3. Mutton
4. Pork (chop, roast)
5. Turkey

height, (127 cm..), are 2,000 calories and the protein requirements are 15 per cent of the total calories.

From table 3 one then reads the formula for a 2,000 calory diet containing 15 per cent of the calories in the form of protein. The formula is: carbohydrate, 60 Gm.; protein, 76 Gm.; fat, 162 Gm. The ketogenic-antiketogenic ratio in the diet will be found to be $\frac{1.5}{1}$

Table 4 shows a diet containing the estimated amounts of the three food ingredients. The menu for 60 on the carbohydrate chart calls for 200 Gm. of 5 per cent vegetables, 100 Gm. of 10 per cent vegetables, 150 Gm. of 10 per cent fruit, 3 Uneeda biscuits and 250 cc. of milk. It will be seen that this menu contains, in addition to the 60 Gm. of carbohydrate, 18 Gm. of protein and 11 Gm. of fat. If 18 is subtracted from 76 and 11 from 162, one will get 58 Gm. of protein and 151 Gm. of fat, respectively. The menu for the latter formula is then read on the protein and fat chart calling for 30 Gm. of cheese, 200 cc. of 40 per cent cream, 2 eggs, 100 Gm. of lean meat or fish, 30 Gm. of butter and 1 Lister bran muffin.

The patient is kept on this diet for one week and the mother is given the following instructions:

1. Directions for collection of the twenty-four hour urine.
2. Directions concerning the use of the food scale (we prefer the Chatillon scale).
3. A written record of the patient's actual food consumption during the two days prior to his return to the clinic.
4. The patient is to return the following week without breakfast.

On the patient's return to the clinic, the following procedure is observed:

1. The patient is weighed.
2. A sample of blood is taken for the determination of sugar and the carbon dioxide combining power.
3. The urine is examined for sugar and ketone bodies. If sugar is present,
4. The carbohydrate, protein and fat contents of the diet are determined from the patient's written record, and the available dextrose and the total caloric value of the diet are calculated. This is an important procedure, for it gives us an accurate record of the patient's actual available dextrose intake. We have observed, from repeated experiences, a marked discrepancy between the diet ordered and the amount consumed.

We increase the available dextrose to a point where glycosuria occurs. By subtracting the amount of dextrose in the urine from the available dextrose in the diet, we determine the amount of dextrose utilized. It has been our experience that when the tolerance is less than 150 Gm., it is desirable to administer insulin in order to assist the utilization of that amount of sugar. We find that when the available amount of dextrose is 150 Gm., we can administer a palatable diet.

If the patient presents glycosuria in the course of the second visit to the clinic, a quantitative estimation of the dextrose excretion is determined in grams. The patient is then ordered to take insulin. The dose of insulin is what Campbell [7] calls "a desugarizing dose." It is equiv-

7. Campbell, W. R., and MacLeod, J. J. R.: Medicine **3**:195, 1924.

alent to 1 unit of insulin for every 2 Gm. of sugar excreted in the urine. Thus, if a patient excretes 40 Gm. of sugar on this test diet, he is given 20 units of insulin in divided doses. We feel that the test diet which is a maintenance diet contains a minimal amount of available dextrose and that any attempt to reduce the dextrose in the diet will necessarily result in either an increase in the ketogenic-antiketogenic ratio or a reduction in the total calories.

If the patient's urine is free from sugar and if there is clinical improvement as evidenced by the fact that he is gaining weight and feels symptomatically well, the carbohydrate is increased and the fat reduced in such proportions as to increase the available dextrose and yet maintain the same caloric requirements. The carbohydrate intake is increased in 20 Gm. increments, and the fat reduced by 10 Gm. If the patient has not gained weight but if his urine is sugar free, the carbohydrates are increased without alteration of the fat in the diet. An attempt is always made to bring the available dextrose in the diet up to the point of the patient's tolerance and at the same time make it sufficiently palatable by reducing the ketogenic-antiketogenic ratio to a more normal balance.

The further management of the diabetic patient who receives insulin depends on his response to insulin therapy. The same fundamental principle is applied as in the description of the management of the patients not receiving insulin, i. e., an attempt is made to have the patient oxidize at least 150 Gm. of available dextrose and to keep the ketogenic-antiketogenic ratio below $\frac{1.5}{1}$ in order to give him a palatable diet. If his urine is sugar-free, the diet is increased or the insulin reduced, depending on whether the diet is adequate or not. If he excretes sugar, the later adjustment of insulin dosage must be modified in each case, and will depend on the judgment of the physician.

It has been found desirable to adjust the diet to meet the whims and appetite of the patient. If the patient wishes more food than our restrictions permit, we do not hesitate to increase the food intake and the corresponding insulin dosage. This must always be done within reasonable limits.

COURSE

Variations in Severity and Tolerance.—The present method for establishing a patient's tolerance consists in determining the degree of glycosuria on a stated test diet, the degree of acidosis, the height of the blood sugar content during fasting in the postabsorptive period, and the nature of the blood sugar response to the oral administration of a definite amount of carbohydrate. Our study of the diabetic children over a period of two and one-half years has enabled us to devise a more satisfactory measure of the patient's dextrose tolerance.

When a patient ingests a stated amount of available dextrose in the diet and excretes a definite amount of dextrose in the urine, then the difference between the two figures represents the amount consumed, whether oxidized or stored. This is a measure of the patient's ability to utilize sugar. When, however, the patient requires a certain amount of insulin to aid in the utilization of the sugar, a more accurate index of the patient's tolerance would seem to be established by determining the ratio of the dextrose utilized to the amount of insulin administered. This ratio we call the dextrose-insulin quotient. Thus, if a patient utilizes 150 Gm. of dextrose with the aid of 10 units of insulin a day, it becomes apparent that his tolerance is far greater than that of a patient who utilizes the same amount of dextrose with the aid of 50 units of insulin. This can be seen more clearly from the dextrose insulin quotient, which is an arbitrary figure and a more concise index. Thus, in the former case, the dextrose-insulin quotient is 15, while in the latter case it is 3. This figure does not necessarily indicate the dextrose equivalent of one unit of insulin, but it does represent the degree of assistance that the patient needs from exogenous insulin.

On this basis, we have made an analytic study of the dextrose-insulin quotients in our patients over successive two and three month periods. This has given us an opportunity to determine any progressive increase or decrease in tolerance under treatment and fluctuating remissions with their causes. These quotients represent averages of determinations made once a week in the outpatient department, and of daily determinations when the patient is in the hospital. Thus, a two month period never represents averages of less than eight determinations.

The question arises as to what part of the variation between subsequent values of the quotient is due to deviations and errors of the factors involved in the calculations. For example, one of the factors in the numerator is the number of grams of available dextrose in the diet. Even when the food consumed is accurately weighed and measured, the quantity of available dextrose in the diet can be estimated only within 15 per cent, which is the approximate variation in the chemical composition of the foods. Other factors influencing the numerator are the accuracy of the scale and measuring instruments, the skill of the attendant in using the scales, the cooperation of the patient in regard to pilfering food, and the accuracy of the value for the total output of urine. The denominator is influenced chiefly by the variations in the potency of the insulin stock, and the care used in administering it.

From a consideration of the various possible sources of error and variations in the chemical composition of food, we estimate that any one quotient may have a possible error of $\pm$ 25 per cent. It would

therefore be unsafe to ascribe clinical significance to a change of less than 50 per cent between two quotients. When, however, quotients are obtained which differ by 100 per cent or more, it appears likely that such are significant of a change in tolerance.

ANALYSIS OF TABLES 6, 7, 8, 9, 10 AND 11

Table 6 (case of Arthur E.) demonstrates a gradual but progressive rise in the dextrose-insulin quotient over a period of five months. The dextrose-insulin quotient rose from 2.5 to 4.5, an increase of 80 per cent. This can be considered outside of the limits of error and reasonably attributed to an improvement in the patient's tolerance.

TABLE 6.—*Case of Arthur E.*

Period	Average Available Dextrose Intake, Gm.	Average Dextrose Excretion, Gm.	Average Dextrose Consumption, Gm.	Average Daily Dosage of Insulin Units	Dextrose / Insulin Quotient
10 days in hospital.....	93.9	12.6	81.9	32.3	2.5
33 days in hospital.....	84.5	2.7	81.8	33.0	2.4
23 days in hospital.....	118.0	4.7	113.3	35.0	3.2
1 month at clinic.....	140.0	23.4	116.6	35.0	3.3
1 month in hospital.....	138.0	6.2	132.8	40.0	3.3
4 weeks at clinic.....	138.0	25.5	112.5	25.0	4.5

TABLE 7.—*Case of Selig C.*

Period	Average Available Dextrose Intake, Gm.	Average Dextrose Excretion, Gm.	Average Dextrose Consumption, Gm.	Average Daily Dose of Insulin, Units	Dextrose / Insulin Quotient	Comment
12 days in hospital	99	8.4	90.6	42	2.2	Nasopharyngitis; temperature, 104
2 months.....	126	10.2	115.8	14	8.3	
1 month.....	147	17.0	130.0	14	9.3	
1 month.....	142	24.3	117.7	18	6.5	Acute infection of upper respiratory tract with temperature of 101.4
3 weeks.....	121	41.0	80.0	20	4.0	Symptoms: gastro-intestinal abdominal pain and vomiting
10 weeks.....	152	21.5	130.5	18	7.2	
10 weeks.....	162	16.4	145.6	20	7.2	
14 weeks.....	162	32.6	129.4	18	7.1	
6 weeks.....	166	16.8	148.2	24	6.2	

Table 7 (case of Selig C.) shows that the dextrose-insulin quotient during an attack of acute nasopharyngitis was 2.2. Then for a period of three months, it varied between 8.3 and 9.3, a definite improvement in tolerance. An acute infection of the upper respiratory tract with gastro-intestinal symptoms brought the quotient down as low as 4, and for a subsequent period of nine months the quotient remained about 7.

Table 8 (case of Yetta K.) demonstrates an interesting phenomenon in the study of the effect of infections on tolerance. While during the first three month period, the dextrose-insulin quotient was 20.5, a

severe attack of scarlet fever brought it down to 7.5 and it has remained at that figure for the past fourteen months without fluctuations. It would seem fairly apparent that an infection of this nature can produce permanent damage to the tolerance.

Table 9 (case of Ida C.) presents data in regard to a patient who was admitted to the hospital in diabetic coma and for the first twelve days of treatment had a dextrose-insulin quotient of 2. The rapid increase in tolerance was illustrated by the fact that during the next twelve days in the hospital, there was a marked rise in the quotient to 8.

TABLE 8.—*Case of Yetta K.*

Period	Average Available Dextrose Intake, Gm.	Average Dextrose Excretion, Gm.	Average Dextrose Consumption, Gm.	Average Daily Dose of Insulin, Units	Dextrose / Insulin Quotient	Comment
3 months.....	116	3.8	111.2	5.4	20.5	
2 months.....	119	37.2	81.8	11	7.1	Attack of scarlet fever
2 months.....	117	2.8	114.2	12	9.5	
2 months.....	111	6.1	105.9	12.8	8.2	
2 months.....	116	3.4	112.6	15	7.5	
2 months.....	128	5.3	122.7	16.2	7.6	
2 months.....	126	8.9	117.1	16.6	7.1	
2 months.....	121	8.3	112.7	15	7.5	

TABLE 9.—*Case of Ida C.*

Period	Average Available Dextrose Intake, Gm.	Average Dextrose Excretion, Gm.	Average Dextrose Consumption, Gm.	Average Daily Dose of Insulin, Units	Dextrose / Insulin Quotient	Comment
12 days in hospital	80	16.0	64.0	31.7	2.0	Diabetic coma
12 days in hospital	121	0.3	120.7	15.1	8.0	
4 months in clinic						
First period.....	150	10.0	140.0	15.0	9.3	
Second period...	140	22.0	118.0	40.0	3.0	Attack of rubella; patient obstreperous; refused several doses of insulin

For the next four months, she was observed during weekly visits to the clinic and her quotient was 9.3. During an attack of rubella, the patient became obstreperous and refused several doses of insulin, and the quotient dropped to 3. This again demonstrates the influence of infection on the sugar tolerance in a diabetic patient.

Table 10 (case of William S.) gives the data for a patient who, over a period of eighteen months while under observation demonstrated no significant variations in the dextrose insulin quotient. During this time the minimum was 6.2 and the maximum 8.7, a difference of 40 per cent. We consider this difference within the limits of deviation due to error and cannot attribute it to changes in tolerance.

Table 11 (case of Hyman P.) presents the data for a patient whose dextrose-insulin quotient during the first 2 months was 3.5. During the last three months, it dropped to 2.2. He suffered with a protracted acute infection of the upper respiratory tract during this last period. The drop in quotient was 60 per cent, significant of a decrease in tolerance.

This method of analysis has given us a criterion for establishing the severity of the diabetes. The lower the quotient, the more severe is the disease. This study also indicates the break in tolerance associated with infections and the determination of the transient or permanent damage that infections can produce. It is interesting to note from table

TABLE 10.—*Case of William S.*

Period	Average Available Dextrose Intake, Gm.	Average Dextrose Excretion, Gm.	Average Dextrose Consumption, Gm.	Average Daily Dosage of Insulin Units	Dextrose / Insulin Quotient
2 months	117	26.2	90.8	14.6	6.2
4 months	135	6.8	128.2	16.6	7.4
2 months	122	13.7	108.3	17.3	7.3
2 months	137	6.2	130.7	18.0	6.2
2 months	158	14.6	143.4	19.2	7.3
2 months	139	16.9	122.1	19.6	6.2
2 months	150	6.7	143.3	16.3	8.7
2 months	148	8.2	139.8	20.0	7.0

TABLE 11.—*Case of Hyman P.*

Period	Average Available Dextrose Intake, Gm.	Average Dextrose Excretion, Gm.	Average Dextrose Consumption, Gm.	Average Daily Dose of Insulin, Units	Dextrose / Insulin Quotient	Comment
2 months	136	28.4	107.6	30.7	3.5	
3 months	117	23.2	93.8	31.2	3.0	
3 months	110	36.3	73.7	32.8	2.2	Acute infection of upper respiratory tract

8 that an attack of scarlet fever has permanently and severely damaged the tolerance of Yetta K. These studies also indicate that there is no clear evidence of progressive improvement in the sugar tolerance of a diabetic child while under dietetic and insulin management. We are compelled to disagree with conclusions reported in the literature that prolonged insulin therapy improves the patient's tolerance.

HYPOGLYCEMIA

The introduction of insulin therapy in the treatment of diabetes has brought to light the new chemical syndrome of hypoglycemia. During the past six years, many data have been gathered on the subject of insulin shock which have revealed its protean nature. Although the first clinical and experimental studies consisted of the observation of a definite train of symptoms of marked hunger, weakness, tremor,

sweats and pallor associated with a low blood sugar, later experiences have added more information to the subject. First, it has recently been demonstrated that the symptoms of insulin shock need not necessarily be attended by a depression in the blood sugar level. Clinical studies by Brown,[8] Leyton,[9] John,[10] Ashe and his co-workers [11] and experimental studies by Shelling and Byron and one of us (W. S. C.) [12] have clearly indicated that the symptoms of hypoglycemic shock can exist when the amount of sugar in the blood is normal or is at an abnormally high level. Leyton reported the blood sugar as high as 250 mg. in a patient with chronic diabetes at a time when all the clinical symptoms of hypoglycemia existed. Ashe and his associates reported a typical hypoglycemic attack with a blood sugar value of 83 mg.

We, too, have observed the same phenomenon in some of our patients, particularly when we determined the blood sugar content early at the onset of the symptoms.

Patient T. K. was admitted to the hospital with all the clinical and laboratory evidence of a severe diabetic acidosis. The blood sugar on admission was 374 mg. per hundred cubic centimeters. While the patient was on a rigid hospital regime of dextrose and insulin, the glycosuria and ketonuria progressively diminished. After one week in the hospital, she suddenly developed the symptoms of hypoglycemia characterized by head sweats, tremors, pallor, hunger and nervousness. Five minutes after the onset of the symptoms, the blood sugar was 105 mg. per hundred cubic centimeters. Administration of 30 Gm. of sugar by mouth caused a disappearance of symptoms in less than five minutes.

Similarly, A. E., in the course of frequent attacks of hypoglycemic shock, revealed blood sugar values of 48, 64, 93 and 178 mg. per hundred cubic centimeters, before the administration of sugar for the complete relief of the symptoms.

On the other hand, patients have been reported to have a blood sugar content as low as 30 mg. without the existence of hypoglycemic symptoms.[13] It therefore becomes apparent that the hypoglycemic state can exist under three conditions: the existence of symptoms and signs with a low or high blood sugar content, and the marked repression of the blood sugar in the absence of symptoms.

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8. Brown, W. L.: Lancet **1**:59, 1924.
9. Leyton, D.: Lancet **2**:1125, 1923.
10. John, H. J.: Diabetes: Statistical Study of One Thousand Cases, Arch. Int. Med. **39**:78 (Jan.) 1927.
11. Ashe, B. I.; Mosenthal, H. O., and Ginsberg, G.: J. Lab. & Clin. Med. **13**:109, 1927.
12. Collens, W. S.; Shelling, D. H., and Byron, C. S.: Am. J. Physiol. **79**:689, 1927.
13. Maddock, S. J., and Trimble, H. C.: Prolonged Insulin Hypoglycemia without Symptoms, J. A. M. A. **91**:616 (Sept. 1) 1928.

The pathogenesis of hypoglycemia is as yet not clearly understood, and an explanation of the phenomenon is open to considerable speculation. While some believe that symptoms result from the rapid reduction of sugar in the blood, others believe that they are due to a rapid disappearance of glycogen from the tissues. Macleod [14] expressed the belief that hyperinsulinemia with the attendant increase in dextrose oxidation in the tissues causes a marked demand on the glycogen reserve of the liver; that the blood acts only as a transporting mechanism for this reserve, and therefore is the last structure to show the reduction in sugar content.

As far as we know, no one has ever observed the state of hypoglycemia at a time when the patient was excreting sugar in the urine. It is therefore reasonable to presume that the only time that the physician or the patient is certain that hypoglycemia does not exist is when glycosuria is present.

The existence of the hypoglycemic state is definitely fraught with danger. The suddeness with which a hypoglycemic convulsion can occur has frequently been reported. Even fatal terminations have been observed.[15] The fact that the hypoglycemic state can exist without the patient possessing subjective symptoms forces us further to stress the importance of avoiding this condition.

One of the most important problems with which we are confronted in the treatment of the ambulatory diabetic child is to arrange the time and dose of insulin administration so as to be of maximum benefit to the patient without causing the appearance of symptoms of hypoglycemia. The difficulty in solving the problem in each patient arises as a result of the fact that juvenile diabetic patients possess a small margin of safety. By that we mean that the period between the existence of hyperglycemia with glycosuria and the development of the hypoglycemic syndrome is frequently not more than two hours. It is as a result of this observation that it has been found more desirable to have the patient excrete a small amount of sugar at all times. We make no attempt to have the urine sugar-free in diabetic children who receive insulin.

Such being the case, the patients or their parents are taught to make fractional examinations for sugar, i. e., every specimen that is voided is examined. A qualitative test is ample, for all that we are interested in is the presence or absence of sugar. This is to guide both them and us in the adjustment of insulin dosage to their diet. It has been found preferable that there shall be at all times small quantities of sugar in the urine, for only then are we certain of avoiding hypoglycemic shock.

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14. Macleod, J. J. R.: Lancet **2**:591, 1923.

15. Brucke, K.: Munchen. med. Wchnschr. **75**:120 and 171, 1928. Beck, W.: Klin. Wchnschr. **6**:212, 1927.

Patients receiving insulin frequently show the cumulative effects, with the development of the hypoglycemic syndrome some time during the evening or night. In these patients, the administration of 100 cc. of orange juice just before retiring has been employed as a prophylactic measure.

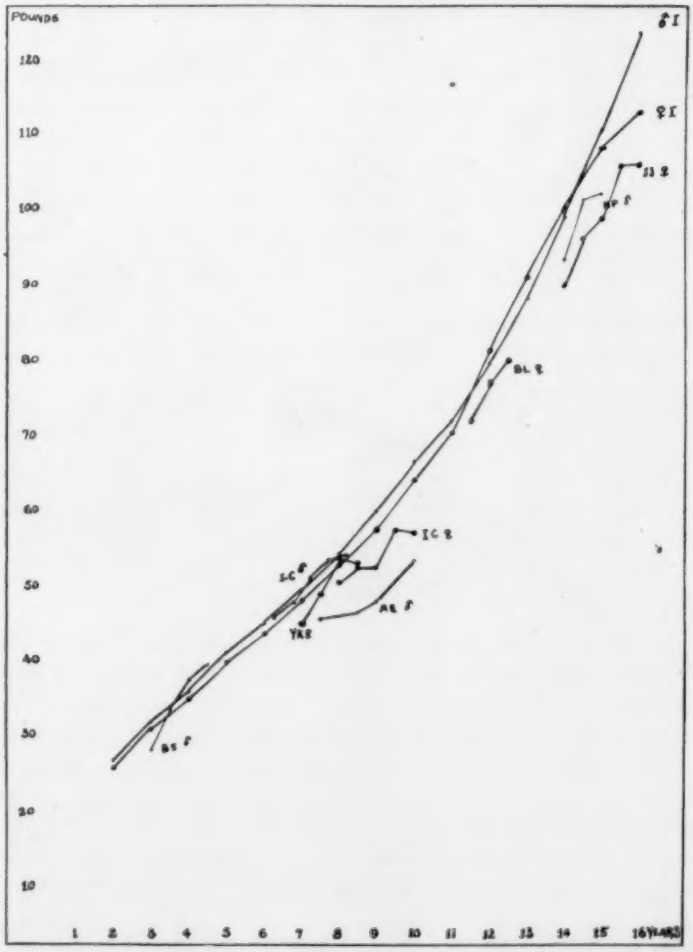

Chart 1.—Weight curve of diabetic children. Standard curves obtained from Abt's Pediatrics, vol. 1, p. 500 (quoting table from Holt and Bowditch). In this chart and chart 2, ♂ *I* indicates the normal male standard and ♀ *I*, the normal female standard.

PERMANENT EFFECTS OF PERSISTENT GLYCOSURIA AND HYPERGLYCEMIA

We have observed that the maintenance of a mild degree of glycosuria is not attended with unfavorable results. This conclusion is based on the records of the patients' growth and weight curves (charts

1 and 2) and on frequent clinical examinations of these patients over a period of two and one-half years. We have never observed any evidence of impairment to any system, particularly the nervous, cardiovascular and renal systems. It will be observed from chart 1 that the rate of increase in weight in all cases follows that of the normal. Chart 2 clearly demonstrates that the rate of growth of diabetic children compared favorably with that of normal children. It is rather interesting

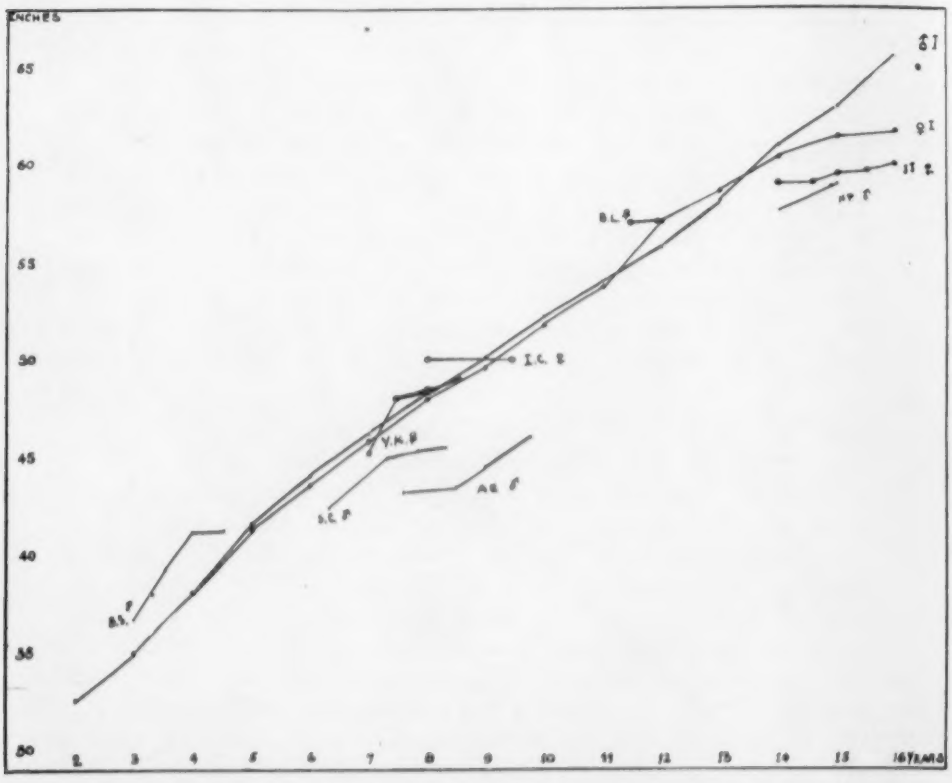

Chart 2.—Growth curve of diabetic children. Standard curves obtained from Abt's Pediatrics, vol. 1, p. 502 (quoting table from Holt and Bowditch).

to note, however, that the rate of growth parallels the normal curve during the first year of treatment, but is slightly retarded thereafter.

As to the undesirable effects of prolonged hyperglycemia on the organism, there seems to be no report in the literature, except a paper by Major and Davis.[16] They presented reports of eight cases show-

16. Major, R. H., and Davis, R. C.: High Blood Sugar with Absence of Sugar in Urine in Diabetes Treated with Insulin, J. A. M. A. **84**:1798 (June 13) 1925.

ing persistent hyperglycemia varying between 250 and 425 mg. per hundred cubic centimeters in diabetic patients under insulin therapy who had been under observation for more than two years. They could discover no evidence of arteriosclerosis, renal disease or any other untoward effects. Attempts to lower the blood sugar in these cases have invariably resulted in the production of attacks of hypoglycemic shock. In controlling these patients, the authors were compelled to be guided entirely by the studies of urinary sugar. This conception is considerably in accord with our own observations of patients who for two years have had persistently high blood sugar values during fasting (table 12).

PERSISTENT ACIDOSIS

Physiologic and clinical investigations in the past have demonstrated a dependence of fat combustion on dextrose oxidation. The works of

TABLE 12.—*Blood Sugar Content of Patients While Fasting During Treatment*

	Patient							
Time	Yetta K.	Selig C.	William S.	Ida C.	Hyman P.	Jennie J.	Arthur E.	Beatrice L.
On admission....	145.0	305.2	350.0	241.4	278.0	255.0	352.0	221.1
3 months.....	130.0	285.4	217.4	110.0	192.0	215.0	207.0	240.8
6 months.....	240.0	258.0	218.0	192.0	325.0	211.2	213.0	178.0
9 months.....	296.0	277.7	270.0	309.0	166.0	269.7	287.0	154.0
12 months.....	220.0	333.0	.....	131.0	.....	212.1	352.6	.....
15 months.....	166.2	300.0	130.8	166.4	.....	240.0	292.6	.....
18 months.....	163.9	349.0	350.0	148.2	.....	347.0	325.7	.....
21 months.....	250.0	246.0	393.0	193.8	.....	227.5	262.0	.....
24 months.....	309.0	280.3	.....	.....	.....	277.6	.....	.....
27 months.....	.....	406.0	.....	.....	.....	235.9	.....	.....

Schaffer [17] and Woodyatt [18] indicate that the oxidation of one dextrose molecule influences the complete oxidation of one molecule of fatty acid. This has been the basis for the presentation by Woodyatt of a safe diet when the ketogenic-antiketogenic ratio is not greater than $\frac{1.5}{1}$. It generally bears out Rosenfeld's original remark that the "fats burn in the fire of the carbohydrates." It has also been our experience that when insulin influences the oxidation of sufficient dextrose in cases of acidosis, the ketone bodies are oxidized and disappear. However, two patients have been observed whose course has not quite fitted the expected rule. The first patient, Yetta K. (table 13), who has been under our observation for two years, has during that entire period demonstrated the presence of acetone in the urine in fairly constant amounts. The carbon dioxide combining power of the blood was normal. This condition has existed in spite of the fact that the

17. Schaffer, P. A.: Medicine **2**:375, 1923.

18. Woodyatt, R. T.: Objects and Method of Diet Adjustment in Diabetes, Arch. Int. Med. **28**:125 (Aug.) 1921.

patient's urine was almost always free from sugar when she was on a diet the available dextrose intake of which varied between 110 and 135 Gm. Under such conditions, one would necessarily suspect that the available ketogenic substances in her diet were so high compared with the available antiketogenic substances that they would account for the acidosis. Over certain periods, we have reduced the ketogenic substances in the diet to an amount when the ketogenic-antiketogenic ratio was as low as $\frac{0.8}{1}$, and yet the condition persisted.

TABLE 13.—*Case of Y. K.*

Date	Carbohydrate, Gm.	Protein, Gm.	Fat, Gm.	Available Dextrose (A.K.)	Ketogenic Substances (K.)	Ratio $\frac{\text{K.}}{\text{A.K.}}$	Insulin, Units	Sugar in Urine, Gm.	Acetone in Urine	Diacetic Acid in Urine	Blood Sugar
11/ 5/27	73	36	112	105	119	$\frac{1.1}{1}$	15	11.2	++	0	245
11/19/27	81	48	122	121	134	$\frac{1.1}{1}$	20	0	+	0	...
12/ 3/27	78	43	112	114	122	$\frac{1.1}{1}$	15	0	++	0	201
12/17/27	76	42	110	112	120	$\frac{1.1}{1}$	15	0	0	0	...
1/ 7/28	75	40	115	110	124	$\frac{1.1}{1}$	15	0	++	0	220
2/18/28	98	48	92	135	107	$\frac{0.8}{1}$	20	0	++	0	...
3/ 3/28	101	48	111	130	124	$\frac{1}{1}$	15	0	++	0	166
5/ 8/28	77	46	105	114	108	$\frac{0.9}{1}$	20	0	0	0	246
6/23/28	88	46	107	125	120	$\frac{0.9}{1}$	10	1.5	++	0	158.5
8/ 4/28	94	46	113	132	125	$\frac{0.9}{1}$	20	5.0	+++	0	...
9/ 1/28	88	44	103	124	115	$\frac{0.9}{1}$	30	2.0	++	0	...

Table 13 illustrates selected periods when the ketogenic-antiketogenic ratio was approximately 1 to 1 or less and the patient sugar free, yet showed the existence of acetonuria. The patient, however, is thriving and shows no untoward effects as a result of the acetone bodies in the urine. She is receiving insulin.

The case of T. K. demonstrates the same phenomenon. She was under our observation for three weeks during which time she was given a sufficiently low fat intake to give a ketogenic-antiketogenic ratio of 1 to 1 and sufficient insulin to keep the urine sugar-free. In spite of the fact that she was sugar-free and the diet was not ketogenic, she showed a constant excretion of acetone in the urine.

COOPERATION

In the course of our contact with the patients and their parents, an attempt is made to educate them to an understanding of some simple fundamental conceptions of the disease. Cooperation is absolutely necessary in the proper management of the child. It is needless to point out the necessity of adherence to the prescribed diet, the proper administration of insulin and the recognition of any untoward manifestations of the disease. The patients and their parents eventually learn to appreciate and properly to evaluate the methods employed in the management of this disturbance. It is interesting to mention that there is a definite age limit within which the patients give us their complete cooperation, i. e., between the ages of 6 and 12 years. It is probably due to the precocity and average high intelligence of the diabetic child. Under the age of 6, it is not expected that the patients will recognize the responsibility of living on a restricted diet, and beyond the age of 12, they seem to establish a careless, adolescent attitude.

SUMMARY

1. The management of the ambulatory diabetic child is described.

2. Tables are incorporated which facilitate the derivation of the diabetic formula and menu.

3. A new method of expressing the sugar tolerance of a diabetic patient in terms of the dextrose-insulin quotient is described.

4. The height and growth rates of diabetic children are reported.

CARDIOSPASM IN INFANCY AND IN CHILDHOOD*

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Cardiospasm in infancy and childhood is seldom mentioned in the literature, and it is practically ignored in the standard textbooks on pediatrics. That it occurs in adults is well known, although frequently it is overlooked. In my experience and that of my colleagues in the Mayo Clinic, it is one of the most common diseases of the esophagus. The question of the etiology of cardiospasm is still debatable, but the preponderance of evidence so far indicates definitely that the condition has a pathologic background. Cardiospasm is regarded by most physicians as a purely functional disorder, old opinions to this effect having been passed on from textbook to textbook without further thought being given to the problem. A statement frequently made is: "Of course cardiospasm is a functional affair as it does not occur in childhood." This statement, and the few but conflicting papers on the subject of cardiospasm in childhood, prompted me to review its frequency as my colleagues in the clinic and I have encountered it in the last few years.

Only those patients were included in the study who gave a clinical history suggestive of cardiospasm and who on roentgenographic examination of the esophagus gave a characteristic picture of cardiospasm. The review included 691 consecutive cases of cardiospasm seen to 1928. For purposes of differentiation, only those cases in which the cardiospasm began before the age of 14 were regarded as having started in childhood. Thirty-four of the 691 patients dated the onset of their symptoms before this age. Twelve of the thirty-four were under 14 years of age at the time they were brought to the Mayo Clinic because of cardiospasm. The tabulation shows the age of onset in the thirty-four cases under consideration.

The three patients in whom the symptoms had begun during the first year of life stated that they never had been able to eat solid food and that they had been informed that considerable difficulty had been encountered with their nursing. The ages of these three patients at the time they were examined at the clinic were respectively 49, 34, and 28 years. All three stated that they never had been able to eat in comfort, until after they had undergone dilatation of the esophagus by means of the hydrostatic dilator.

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* From the Division of Medicine, The Mayo Clinic.

The duration of symptoms in the whole group of thirty-four patients varied from less than one to forty-nine years, with an average of twelve and six-tenths years. The general average is greatly increased owing to the fact that six of the patients had had symptoms for twenty-eight years or longer.

The mode of onset of symptoms, except in those cases which had begun in infancy, was the same as that reported by adults. It becomes evident from the study that the onset is usually insidious or that the disease may be noticed first when the child is recovering from some acute infection of childhood, such as measles or whooping cough. The first symptom usually is dysphagia; some solid particle of food lodges in the esophagus and is either regurgitated or is washed down with water. The child soon learns to take several swallows of solid food and then to force them down by rapidly drinking a glass of water. Generally, some of

Age of Onset in Thirty-Four Cases of Cardiospasm in Each of Which the Disorder Began Before the Age of Fourteen

Age at Onset, Years	Number of Cases
1	3
2	..
3	1
4	1
5	1
6	4
7	3
8	2
9	6
10	1
11	3
12	4
13	5
Total	34

the water is retained in the esophagus after the food passes through and must be regurgitated. At times, after the first few mouthfuls have been forced through, the child may be able to consume the remainder of the meal without difficulty. Liquids usually give as much difficulty as solids. Although the patient may be able to force solid food into the stomach by drinking large quantities of water, still he may be unable to force down water of itself. Cold fluid usually gives as much trouble as any type of solid food. The dysphagia may fluctuate in severity from day to day. With some meals the esophagus may offer little trouble, whereas, on the following day, it may close completely, allowing nothing to pass through, and may remain closed for from a few minutes to several days.

The food regurgitated is usually in the same condition as when it was swallowed, and is not mixed with gastric juice. Usually a considerable amount of mucus is mixed with the regurgitated material, for the secretions accumulate in the dilated esophagus. Drooling of material

from the mouth, on lying down at night, with soiling of the pillow, is a frequent complaint.

Pain is a variable symptom. When present, it frequently is sharp, is felt under the lower end of the sternum, and is especially noticeable when food passes through the cardia into the stomach. The severity of the pain varies considerably. It may be simply a sense of discomfort, or it may be severe enough to cause the patient to double up. Of the thirty-four patients studied in this series, fourteen complained of pain.

In very young children, the onset of the symptoms may be such that the condition may be mistaken for croup or for some pulmonary disease. On lying down at night, there is coughing and strangulation, due to the food and fluid which was retained in the esophagus welling over into the larynx. The child may receive treatment for bronchitis for a long period of time before the true nature of the disease is discovered. Occasionally, as in adults, bronchiectasis or pulmonary abscess may develop due to inspiration into the bronchi of regurgitated material. When this occurs, the true cause of the symptoms may be completely overlooked, and the prognosis is modified.

Although most children complain of the food lodging under the lower end of the sternum, an occasional patient may feel that the point of obstruction is in the throat or under the middle of the sternum. This probably accounts for the tonsillectomies that were performed for the relief of symptoms in many cases.

The loss of weight is variable. Some patients lose practically no weight, whereas others show definite loss. There is usually no change in the percentage of hemoglobin or in the blood pressure.

The treatment for cardiospasm in infancy and childhood is the same as that for the same condition in adult life. The hydrostatic dilator is employed. Unless the esophagus is completely obstructed or the child is markedly dehydrated, it is given a twisted silk thread, size D, to swallow. That a twisted thread is used is emphasized, for it is much more difficult to swallow braided thread. The patients are instructed to swallow about 5 yards of thread and to take it slowly, not faster than 1 foot an hour. They are told, also, not to chew the thread, as this tends to product snarls and knots. They are permitted to eat their regular meals during this time, although the amount usually is restricted. The patients usually require from twenty-four to seventy-two hours to swallow the necessary length of thread; they sleep with it in place. The thread serves a twofold purpose: 1. If used correctly, it prevents the operator's punching a hole through the wall of the esophagus when he is passing sounds. 2. It acts as a most efficient guide when sounds and dilators are being passed through the cardia. When the size of the sac above the cardia is appreciated, the difficulty and the danger of trying to introduce a sound into the stomach without a guide are readily under-

stood. The thread usually is anchored so firmly in the intestine that it tends to break when an attempt is made to pull it out.

In the management of infants and very young children who cannot co-operate, the following routine is observed. The child is placed in a hospital and a special nurse is assigned. It is the nurse's duty to put the thread in the infant's mouth and to try to get the baby to swallow it. Contrary to expectations, the child soon swallows the thread rather than continuing to spit it out. If children are extremely obstinate, the thread is introduced through the nose. If they are dehydrated, proctoclysis is employed during this period. It has been found that children will swallow the thread sooner for a nurse than for their parents. In our experience, children usually cause less trouble than older persons, who frequently think they know best how to do it and do not listen to instructions.

If any difficulty is expected in performing the dilatation, a thread of size EE is tied to the thread of size D and is allowed to enter the intestines. Thread of size EE is stronger than that which generally is used and is less likely to break. A sound, size 41 French, is first passed through into the stomach. This discloses the approximate site of the cardia and likewise permits of better drainage of the esophagus. The benefit from this procedure is, however, of only short duration, and it is usually deemed advisable to use the hydrostatic dilator in a few days. A hydrostatic dilator somewhat smaller than the size used for adults is used for children, and the usual technic for hydrostatic dilatation is carried out.

Anesthesia is not used, since the discomfort of the patient serves as an indication of whether or not one is definitely dilating the cardia. It is a good rule to remember that if the patient does not have pain under the lower end of the sternum at the time of dilatation, generally the cardia has not been satisfactorily dilated. Furthermore, the use of anesthesia is contraindicated for the reason that during dilatation there is practically always some regurgitation of the mucus and the food which has been retained in the esophagus and this gives rise to the danger of aspiration of regurgitated material into the lung, with resulting pneumonia or pulmonary abscess. The fluoroscope may be used to guide an opaque dilator into the cardia. This method, however, has disadvantages: the work must be done in a dark room, with the usual associated confusion; furthermore, it is not always possible definitely to locate the cardia without using a suspension of barium, and this, by adding to the material to be regurgitated, causes the patient greater discomfort.

The results obtained by means of the hydrostatic dilator in the treatment of infants and children with cardiospasm have been satisfactory. All patients except two were treated by this method. One patient was in a moribund condition at the time she arrived at the clinic and she

died before treatment of any sort could be instituted. Death was found to be due to inanition and bronchopneumonia. The other patient was unable to remain for treatment. Of the thirty-two patients treated, twenty-five have had excellent results up to the time of writing. No record was at hand as to late results in the seven remaining cases, although all seven patients apparently were completely relieved at the time of their dismissal.

CONCLUSION

Cardiospasm in childhood is of relatively greater frequency than it is generally supposed to be. The hydrostatic dilator offers the most satisfactory and safest method of treatment.

BASAL METABOLISM IN CHILDREN OF ABNORMAL BODY WEIGHT *

II. IN UNDERWEIGHT CHILDREN

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In a previous paper,[1] we presented the results of basal metabolism tests on seventy overweight, but otherwise normal, children. These results seem, in the main, to agree with those found in overweight adults, and afford further proof that the basal metabolic rate in the condition of overweight, unless associated with other pathologic conditions, is usually normal, with a tendency toward high normal rate, owing to the stimulus afforded by a condition of plethora in the cells.

In this paper, we present our observations in a similar series of seventy underweight, but otherwise normal, children. We tried, as far as possible, to exclude those children in whom some definite pathologic condition was present which might affect the basal metabolic rate. We include thirty-five boys and thirty-five girls who were sent to our metabolism clinic because of "chronic malnutrition." Their ages ranged from 5 to 15 years. The deviation in weight ranged from 10 to 33 per cent less than normal.

The Krogh calorimeter was used, and the usual accepted technic adopted as to postabsorptive state and initial rest period. Four determinations of five minutes each—in some cases more than four determinations were made on each child, the lowest being accepted as the true basal metabolic rate. We tried, as far as possible, to exclude all disturbing factors such as discomfort, excitement, muscular movement, surreptitious ingestion of food, rise in temperature, etc., which might cause a temporary nonbasal elevation in metabolism.

Tables 1 and 2 record the essential data in all cases. Age was recorded from the nearest birthday. The standing height of the subject was measured without the shoes on. Weight was recorded with the subject dressed in indoor clothing but without shoes on. The condition of nutrition was measured by the Pirquet pelidisi,[2] by the Baldwin-Wood tables [3] and by the Pirquet-Camerer [4] chart.

* Submitted for publication, Jan. 31, 1929.

* From the Pediatric Service of Mount Sinai Hospital, Bela Schick, Chief of Staff.

1. Topper, Anne; and Mulier, Hannah: Basal Metabolism in Overweight Children.

2. Von Pirquet, C.: System der Ernahrung, Berlin, 1919, part 2, p. 284.

3. Baldwin and Wood: Weight-Height-Age Tables, Mother and Child, July, 1923.

4. Von Pirquet, C.: Ztschr. f. Kinderh., 1913.

TABLE 1.—*Essential Data Obtained in Series of Underweight Boys*

Case Number	Age	Sitting Height, Cm.	Standing Height, Cm.	Weight, Kg.	Pulse Rate	Nutritional State: Pirquet Pelidisi	Nutritional State: Per Cent Deviation, Baldwin-Wood	Nutritional State: Per Cent Deviation, Pirquet-Camerer	Heat Production, Total Calories for 24 Hours: Observed	Pirquet Prediction	Per Cent Deviation	Talbot Prediction *	Per Cent Deviation	Remarks
1	11	70	130	26	72	91	10	13	984	980	— 0	1,045	— 5	
2	11	70	127	25	70	90	10	12	1,056	980	+ 7	1,015	+ 4	
3	10	70	130	26	72	91	10	13	908	980	+ 1	1,045	— 4	
4	10	72	130	26	68	88	10	13	956	1,037	— 7	1,045	— 8	
5	11	73	135	29	70	90	10	11	1,117	1,065	+ 4	1,105	+ 1	
6	9	69	126	24	80	90	10	14	999	952	+ 5	1,000	0	
7	10	70	130	26	72	91	10	13	908	980	+ 1	1,045	— 4	
8	9	69	126	24	72	90	10	14	999	952	+ 5	1,000	0	
9	9	68	123	24	68	91	10	10	876	924	— 5	965	— 9	
10	6	65	115	19	70	90	11	17	785	844	— 7	875	—10	
11	13	74	138	30	94	90	11	12	1,250	1,095	+14	1,140	+ 9	
12	12	75	140	31	84	90	12	11	1,248	1,125	+ 8	1,165	+ 7	
13	11	72	133	27	64	90	12	14	1,000	1,036	— 3	1,083	— 7	
14	12	72	136	28	70	91	14	15	968	1,036	— 6	1,072	—10	
15	9	69	127	24	80	90	14	16	1,000	952	+ 5	1,015	0	
16	7	64	115	19	70	90	14	17	864	815	+ 5	875	0	
17	12	74	137	29	68	89	15	13	1,052	1,095	— 6	1,130	— 8	
18	13	81	158	42	88	92	17	10	1,480	1,312	+12	1,377	+ 8	Palpable thyroid; no symptoms
19	13	83	165	45	72	92	17	..	1,344	1,377	— 2	1,468	— 8	
20	15	85	165	47	72	91	18	..	1,248	1,444	—13	1,468	—15	Same subject as in case 18; thyroid still palpable
21	10	69	127	22	80	87	18	23	998	952	+ 4	1,015	— 1	
22	11	72	135	26	72	89	18	20	998	1,036	— 3	1,105	—10	
23	6	60	108	15	76	88	19	25	784	720	0	820	— 8	
24	7	65	115	18	68	87	19	21	794	845	— 6	875	— 9	
25	7	64	115	18	70	88	19	21	792	843	—10	875	— 9	
26	11	76	140	28	68	86	20	20	1,104	1,155	— 4	1,165	— 5	Palpable thyroid; no symptoms
27	9	69	127	21	70	86	20	24	888	952	— 7	1,015	—12	
28	14	78	143	30	84	86	20	18	1,362	1,218	+12	1,200	+13	
29	8	67	123	19	54	85	25	24	796	897	—11	965	—17	
30	9	68	125	20	60	86	27	27	816	924	—12	990	—17	
31	6	64	112	15	70	84	27	30	720	815	—11	850	—14	
32	12	72	132	22	54	84	29	29	904	1,036	—12	1,072	—15	
33	9	68	121	19	60	85	31	26	816	924	—11	945	—13	
34	10	72	132	21	60	82	31	30	888	1,036	—14	1,072	—17	
35	9	72	130	20	68	81	31	30	820	980	—16	1,045	—21	

* Projected beyond 150 Cm.

TABLE 2.—*Essential Data Obtained in Series of Underweight Girls*

Case Number	Age	Sitting Height, Cm.	Standing Height, Cm.	Weight, Kg.	Pulse Rate	Nutritional State: Pirquet Pelidisi	Nutritional State: Per Cent Deviation, Baldwin-Wood	Nutritional State: Per Cent Deviation, Pirquet-Camerer	Heat Production, Total Calories for 24 Hours: Observed	Pirquet Prediction	Per Cent Deviation	Talbot Deviation *	Per Cent Deviation	Remarks
1	10	70	131	26	68	91	10	10	972	980	0	975	0	
2	8	67	125	23	62	91	12	11	864	897	— 3	915	— 5	
3	5	57	100	14	80	91	13	14	670	649	+ 3	675	0	
4	10	71	134	27	60	91	13	10	898	994	— 9	1,005	—10	
5	12	78	152	37	90	92	14	12	1,338	1,212	+12	1,183	+15	Palpable thyroid; no symptoms
6	11	78	148	32	68	89	14	17	1,248	1,216	+ 3	1,148	+ 8	
7	10	72	137	27	68	90	15	13	936	1,036	— 9	1,037	— 9	
8	10	71	137	28	80	92	15	10	1,104	1,108	0	1,037	+ 6	
9	10	72	133	26	60	88	15	10	960	1,036	— 7	995	— 3	
10	10	70	132	25	60	89	15	12	888	990	—10	985	— 9	
11	11	77	144	32	66	89	16	10	1,142	1,185	— 3	1,108	— 2	
12	11	73	137	27	72	88	16	13	998	1,065	— 6	1,037	— 3	
13	11	75	144	33	74	92	16	10	1,208	1,140	+ 7	1,108	+10	
14	11	77	150	36	60	92	16	10	1,073	1,185	— 9	1,160	— 7	
15	8	67	120	21	70	89	16	12	816	897	— 9	866	— 6	
16	10	68	124	22	70	89	17	13	873	924	— 5	904	— 3	
17	9	68	128	23	60	90	18	14	840	924	— 9	945	—10	
18	11	75	141	29	68	88	19	13	1,076	1,124	— 4	1,078	0	
19	12	78	154	37	88	92	19	16	1,368	1,212	+12	1,207	+13	Palpable thyroid; no symptoms
20	11	77	150	34	60	90	20	15	1,137	1,185	— 4	1,160	— 1	
21	11	78	148	33	64	89	20	15	1,214	1,216	0	1,148	— 5	
22	14	82	158	40	88	90	20	17	1,536	1,344	+14	1,275	+20	
23	13	80	154	36	90	89	20	18	1,532	1,312	+17	1,206	+26	
24	6	60	115	17	70	91	21	22	692	720	— 4	817	— 9	Palpable thyroid; no symptoms
25	8	66	126	20	80	89	22	24	904	871	+ 3	925	0	
26	14	82	158	39	76	89	23	19	1,252	1,344	— 5	1,250	0	Same subject as in case 19; menses established for 10 months
27	12	79	152	34	90	89	23	18	1,488	1,266	+18	1,183	+25	
28	11	72	141	27	80	89	23	21	1,005	1,036	— 3	1,078	— 6	
29	8	65	122	18	68	87	25	25	741	844	—12	885	—15	
30	12	72	141	27	88	89	25	20	1,128	1,036	+ 9	1,078	+ 5	
31	14	77	152	34	86	91	26	20	1,280	1,185	+ 7	1,183	+ 8	Menses established for 1 year
32	10	72	139	25	64	87	27	22	924	1,036	—11	1,057	—12	
33	10	68	130	21	60	87	28	24	800	924	—13	965	—17	
34	11	75	140	25	60	84	29	23	936	1,125	—16	1,068	—12	
35	11	77	149	29	60	86	33	28	984	1,185	—17	1,158	—15	

* Projected beyond 138 Cm.

Table 3 summarizes the essential data by age and sex.

Here again, as in our series of overweight children, we used as prediction standards only those which are based on a linear formula. Any other formula depends on the weight of the child; consequently, the relative proportion of active and inactive tissue affects the final results, and in cases in which the body weight deviates from the normal, such formulas would obviously be useless.

We used the Pirquet sitting-height standard,[5] which we find the best single prediction standard in all cases. Benedict[6] recently recommended that between the ages of 1 week and 12 years, basal metabolism results should be referred to height alone. Accordingly, we used the Talbot[7] standard based on height for comparison with the Pirquet standard. We found the two standards in accord within 5 per cent in most cases.

TABLE 3.—*Basal Metabolism of Underweight Children: Summary by Age and Sex*

	Boys				Girls					
Years	Normal Basal Metabolic Rate	Below — 10	Above + 10	Totals	Normal Basal Metabolic Rate	Below — 10	Above + 10	Totals	Grand Totals	Years
5	..	..	..	..	1	..	..	1	1	5
6	2	1	..	3	1	..	..	1	4	6
7	3	..	..	3	..	..	..	..	3	7
8	..	1	..	1	3	1	..	4	5	8
9	5	3	..	8	1	..	..	1	9	9
10	4	1	..	5	7	2	..	9	14	10
11	6	..	..	6	8	2	..	10	16	11
12	3	1	..	4	2	..	3	5	9	12
13	1	..	2	3	..	..	1	1	4	13
14	..	..	1	1	2	..	1	3	4	14
15	..	1	..	1	..	..	..	..	1	15
Totals...	24	8	3	35	25	5	5	35	70	

In chart 1, we plotted the basal metabolic rate as measured by the von Pirquet standard. In chart 2, we showed, similarly, the basal metabolic rates according to the Talbot standard.

Chart 3 shows the relationship between body weight and basal metabolism. The upper curve shows the variation of each child from his normal basal metabolic rate. The lower curve is correspondingly plotted to show the percentage of deviation from the normal weight of each child, beginning with 10 per cent underweight and increasing to 33 per cent underweight.

LITERATURE

We found little in the literature on basal metabolism in malnutrition, and most of what we did find, concerned infants and adults.

5. Von Pirquet, C.: An Outline of the Pirquet System of Nutrition, Philadelphia, W. B. Saunders Company, 1922.

6. Benedict, F. G.: Am. Phil. Soc. **43**:25, 1924; Chem. Abstr., vol. 18, p. 3640.

7. Talbot, F. B.: Basal Metabolism of Children, Physiol. Rev. **5**:477 (Oct.) 1925.

In malnourished adults, most investigators have found a lowering of the basal metabolic rate. This phenomenon was recognized at an early date. Magnus-Levy,[8] in 1906, first called attention to it. The observation was later corroborated by Benedict and Joslin,[9] Allen and DuBois,[10] Zuntz and Loewy,[11] Roth and Smith [12] and Lusk.[13] All these workers found a reduction in the basal metabolic rate in the condition of under-

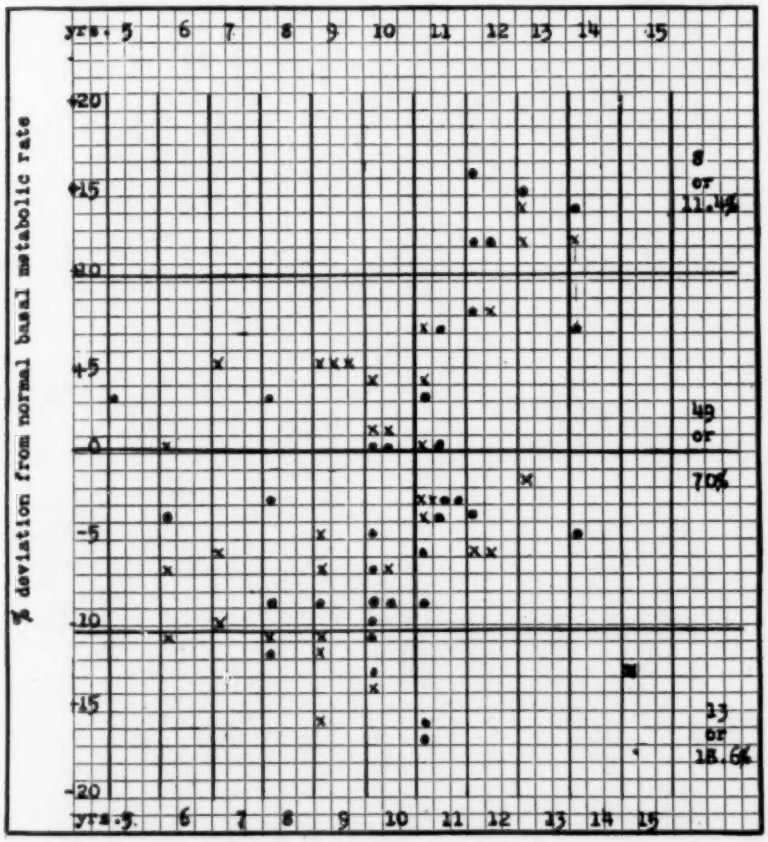

Chart 1.—Basal metabolism of underweight children according to the Pirquet standard; *x* indicates boys, the black circle, girls.

8. Magnus-Levy: Ztschr. f. klin. Med. **60**:177, 1906.

9. Benedict and Joslin: Publication 136, Carnegie Inst. of Washington, 1910; publication 176, Carnegie Inst. of Washington, 1912.

10. Allen, F. M., and DuBois, E. F.: Clinical Calorimetry, Arch. Int. Med. **17**:1010 (June) 1916.

11. Zuntz and Loewy: Biochem. Ztschr. **90**:244, 1918.

12. Miles, Roth and Smith: Publication 280, Carnegie Inst. of Washington, 1919.

13. Lusk: Physiol. Rev. **1**:523, 1921.

nutrition. We may say that all results agree—there is a lowering of the basal metabolic rate associated with the condition of underweight in adults.

The earliest studies of infantile malnutrition were made by Fleming,[14] later studies by Talbot.[7] The total heat production of a large proportion

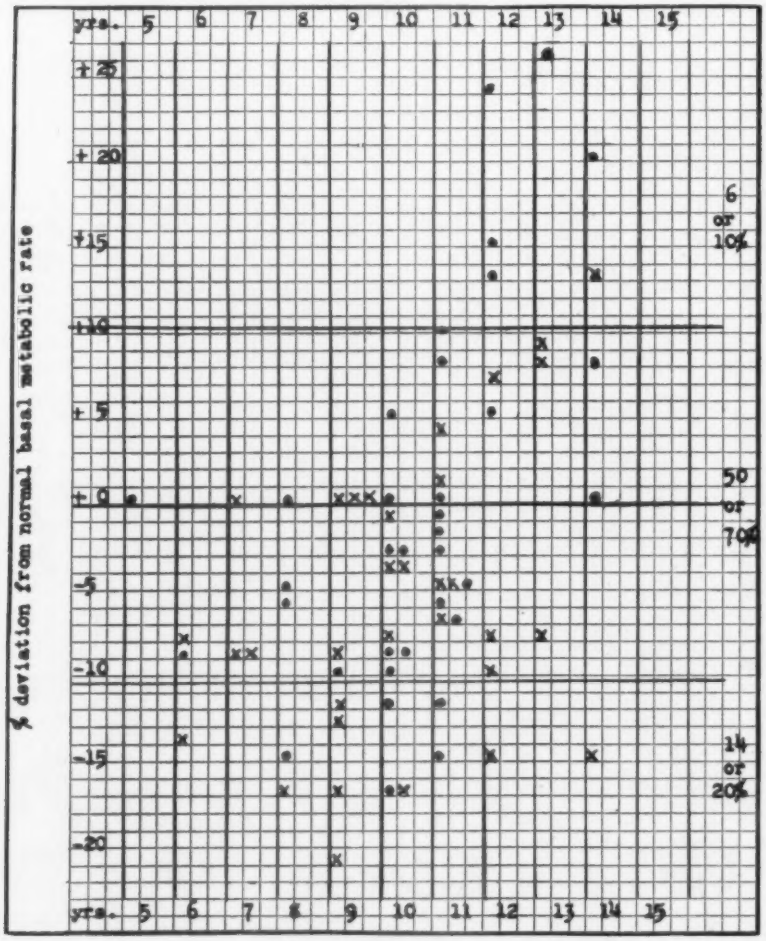

Chart 2.—Basal metabolism of underweight children according to the Talbot standard; *x* indicates boys, the black circle, girls.

of these infants was distinctly less than that of normal children. In summing up the results, Talbot said "marked undernutrition may result in a metabolism which is seemingly elevated, per unit of body mass, but the total heat production per twenty-four hours is actually lower."

14. Fleming, G. B.: Metabolism in Infantile Atrophy, Quart. J. Med. **14**:171, 1921.

There are only two large series of studies reported on older underweight children. Blunt and her co-workers[15] reported a series of twenty-eight children, of whom only twenty-two were 10 per cent and more underweight. They found an increased basal metabolic rate per kilogram of body weight, and per square meter of body surface. But as both these standards take body weight into consideration, we consider them inapplicable in cases in which body weight deviates from the normal. We could not compare these results with our own, as the sitting heights of the children were not included in the data. Recalculated, however, from their height alone, the total heat production was actually slightly low.

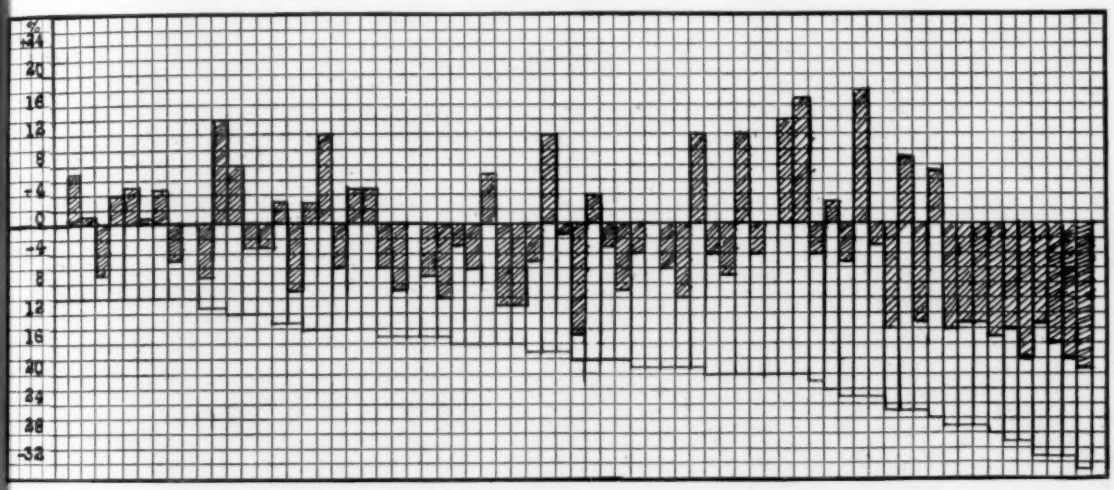

Chart 3.—Basal metabolism of underweight children; upper curve (shaded part) shows the percentage of deviation from the normal basal metabolism of each child; lower curve (unshaded part) shows, correspondingly, the percentage of deviation from the normal weight of each child.

The other series of basal metabolic studies in underweight children was reported by Wang, Kern, Frank and Hays.[16] They made tests on forty-one children, seventeen of whom were 10 per cent and more underweight. They reported that "there seems to be a tendency toward a lower metabolism with increasing underweight as shown especially by the age and height standards."

These two series are the only studies comparable with ours. Our series is the largest we could find in the literature.

15. Blunt, Nelson and Oleson: J. Biol. Chem. **49**:247, 1921.

16. Wang, C. C.; Kern, R.; Frank, M., and Hays, B. B.: Metabolism of Undernourished Children, Am. J. Dis. Child. **32**:350 (Sept.) 1926.

RESULTS AND COMMENT

Chart 1 shows that forty-nine children, or 70 per cent of all cases, had a basal metabolic rate within the normal range of from plus to —10. Eight children, or 11.4 per cent, had a basal metabolic rate above + 10, while thirteen, or 18.6 per cent, had a basal metabolic rate below —10. When measured by the Talbot prediction standard, these results are practically the same (chart 2). In other words, in the present fairly large series of underweight children, the majority did not show any considerable deviation from a normal basal metabolic rate.

We found the same to be true of our series of overweight children, in which 71 per cent had a normal basal metabolism. In our overweight series, we had no cases in which the basal metabolic rate was below —10. The general tendency was toward a high normal rate. This is what one would expect in most cases of overnutrition. It is now pretty generally known that the condition of overnourishment is accompanied by an increased basal metabolic rate, or a luxury consumption of energy. Atkinson and Lusk [17] and Helmreich and Wagner [18] have demonstrated an increase in basal metabolic rate following a course of overfeeding, an increase which persists after the course of overfeeding.

In our cases of overweight, while the general tendency was toward a high normal basal metalic rate, there was no definite relationship between increased weight and increased metabolic rate. When we analyze our underweight series, we see from chart 3 that there were twenty-two cases in which the basal metabolic rate rose above the zero line, while in forty-eight cases it fell below the zero line. In other words, the majority of our underweight children had a tendency to a lowered basal metabolic rate. We noted also that, in general, decreasing body weight was associated with a lowering of the metabolism, and that when the subject was more than 25 per cent underweight, the basal metabolic rate fell even below the lowest normal limit of —10.

Talbot said that "no metabolism values for youth may be considered as under basal; the tendency always being for such values to be above rather than below the true basal." We may, therefore, consider our low results in this series as truly basal. Our results point to a lowered metabolic rate associated with the underweight state in children. This lowering of the metabolic rate may be a definite compensatory economy of oxidation which probably tends to protect the organism from the evil results of starvation. There is a gradual adaptation downward of the basal metabolic rate to a long continued decreased intake of food, and, in the majority of cases, an increased caloric intake will cause a

17. Atkinson and Lusk: J. Biol. Chem. **41**:13, 1919.

18. Helmreich and Wagner: Ztschr. f. Kinderh. **38**:206, 1924.

rise in the basal metabolic rate. Anderson and Lusk,[19] in experiments on dogs, found a persistently lowered basal metabolism after a period of fasting. Helmreich,[20] too, believed that the height of the basal metabolic rate depends on the previous intake of food.

If there is a compensatory lowering of the metabolic rate in a condition of undernutrition, how may we account for the children who did not have a lowered metabolic rate? How account especially for those cases in which the basal metabolic rate was above the upper normal limit of +10?

We tried to eliminate all those conditions which might cause a temporary rise in metabolic rate. We made at least four tests on each child. Clinically, there were no evidences of hyperthyroidism. (Three of these children did have a palpable thyroid gland. These cases will be discussed later.)

It is possible that in a few cases, there was still a nonbasal, temporary increase in metabolic rate, and this factor can never quite be disregarded in a consideration of metabolism tests in children, especially younger children. However, among chronically undernourished children, we must recognize the type of child who is restless, overactive and nervous, who consumes large amounts of food and who still does not gain weight. This is the child who is most likely to show a high basal metabolic rate in spite of extreme underweight. While some factor may be working in the direction of a compensatory lowering of his metabolic rate, some other factor is at work which increases it. This may be an endocrine factor. Cases are commonly observed in which a plump infant, without demonstrable cause, suddenly becomes thin and undernourished, with hyperactivity and a decreased intake of food and, conversely, cases in which an underweight child suddenly becomes fat. Some endocrine factor may also be at work in all these cases. And so it may be with the markedly underweight child who still has a high basal metabolic rate. This type of child would, perhaps, benefit more by prolonged rest than by an increased caloric intake.

It was interesting to note that eight of our subjects had a basal metabolic rate above the upper limit of the normal +10 per cent. While we cannot draw conclusions from eight cases, it seems to us significant that these eight children were all between 12 and 14 years of age. This, the period of pubescence, is associated with many important changes in the child. There is an increase in growth, an increased activity of the glands of internal secretion and an awakening of sexual life, all of which probably influence the basal metabolic rate. In our overweight children, we also found a high basal metabolic rate at this

19. Anderson and Lusk: J. Biol. Chem. **32**:421, 1917.

20. Helmreich, E.: Biochem. Ztschr. **146**:153, 1924.

age period. We would like to point out again that other investigators have found an increase in the basal metabolism at puberty. DuBois,[21] Janet,[22] Lax and Petenyi,[23] Göttche[24] and Bedale[25] all found an increased basal metabolic rate in normal children between the ages of 11 and 14. In our overweight children, we found an increased metabolic rate at this period, an increase which occurred earlier in the girls than in the boys. In our present series of underweight children, we also found a small number who, in spite of being markedly underweight, showed an increase in the basal metabolic rate at this time. Three of these children had a palpable thyroid gland with no symptoms of increased thyroid function. In the other five, the thyroid gland was not palpable. In none of our overweight children could we palpate the thyroid gland. We believe that these facts point to a possible temporary increase in the function of the endocrine glands at the period of puberty, which may be accompanied by a palpable thyroid gland, but which may not be associated with any other sign than a temporary increase in the basal metabolic rate. Should further investigation confirm this observation in normal children, it would explain the so-called "hyperthyroidism" of puberty which is not an uncommon clinical phenomenon.

SUMMARY

We present the results of basal metabolism tests made on seventy underweight children. The outstanding observations are, briefly:

1. The majority (70 per cent) of these children had a basal metabolic rate which came within the usual normal limits of plus and minus 10 per cent. The tendency was toward a low normal.

In a similar series of seventy overweight children, the majority also had a normal basal metabolic rate, the tendency, however, being toward a high normal.

2. In subjects becoming increasingly underweight, there was an increased lowering of the basal metabolic rate. In most cases, when the subject was more than 25 per cent underweight, the basal metabolic rate fell below the lowest normal limit of minus 10 per cent.

In our overweight series, there was no association between the overweight percentage and increased basal metabolic rate.

3. Eight markedly underweight children had a basal metabolic rate above the upper normal limit of plus 10 per cent. These children

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21. DuBois, E.: Basal Metabolism in Health and Disease, Philadelphia, Lea & Febiger, 1927, p. 227.

22. Janet, J. méd. franç., June, 1923, vol. 12.

23. Lax and Petenyi: Monatschr. f. Kinderh. **36**:381 (Sept.) 1927.

24. Göttche: Monatschr. f. Kinderh. **32**:22, 1926.

25. Bedale: Proc. Roy. Soc., London **94**:368, 1923.

were all between 12 and 14 years of age—the important period of pubescence, with which are associated marked physical, physiologic and sexual changes.

Among our overweight children, also, an appreciable number had an abnormally high basal metabolic rate at this same age period.

CONCLUSIONS

From the results of our basal metabolism tests made on seventy underweight children and on seventy overweight children, we conclude that:

1. The majority of overweight, but otherwise normal, children have a normal basal metabolic rate, with a tendency toward a high normal.

The majority of underweight, but otherwise normal, children have a normal basal metabolic rate, with a decided tendency toward a low normal. In children becoming increasingly underweight, there is a greater lowering of the basal metabolic rate. When the underweight percentage falls to below 25 per cent, the basal metabolic rate falls below the lowest normal limit of minus 10.

Basal metabolism depends on the nutritional condition of the cells, plethora acting as a stimulant and, conversely, inanition acting as a depressant to the basal metabolic rate—a mechanism, to a certain extent, compensatory for preventing excess of fat in the former case, and staving off starvation in the latter.

2. At the period of puberty, between the ages of 12 and 14 years in girls and somewhat later in boys, a certain percentage of children, irrespective of whether the body weight is above or below normal, there is probably a temporary increase in function of the thyroid gland, which may be associated with no other sign than an abnormally high basal metabolic rate.

Individual studies at short intervals during this important age period are desirable.

ORGANISMS ASSOCIATED WITH COMMERCIALLY PREPARED INFANT FOODS*

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Recently there has been considerable discussion among pediatricians and laboratory workers as to the sanitary quality of certain of the prepared infant foods appearing on the market. In 1927, G. F. Dick and Gladys H. Dick [1] reported the presence of hemolytic or *viridans* types of streptococci in four out of five samples of powdered milk preparations which they examined and concluded that the methods of manufacture do not destroy the organisms carried in the milk previous to the manufacturing process. They suggested that the use of powdered milk without previous boiling or pasteurization may lead to difficulties.

The present study has been undertaken in connection with a more extensive investigation on the bacteriology of prepared infant foods to determine the prevalence of hemolytic streptococci in such foods in order that those who are using them may be enlightened as to their sanitary qualities and that manufacturers may better know the actual bacterial content of their products as they reach the infant.

Samples of the products of the various manufacturers who have made available prepared infant foods were secured on the open market. Both protein milks and carbohydrate modifiers as well as certain specially reconstituted mixtures have been studied. The results presented deal only with the distribution of hemolytic streptococci in such products.

Unopened cans of the products were obtained, and 1 Gm. of the contents was weighed aseptically into 100 cc. of water from which the required dilutions were made. Plates were prepared, a veal infusion agar, to which had been added red blood cells from the sheep or the goat, being used. The veal infusion agar was prepared by infusing 500 Gm. of lean veal in 1 liter of water and after the usual clearing and adjustment of reaction to p_H 6.8 to 7.0, 5 Gm. of sodium chloride, 5 Gm. of Difco peptone, and 15 Gm. of agar were added per liter of broth. Previous to plating, red blood cells from the horse or the goat were added to this medium.

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* Submitted for publication, March 15, 1929.
* From the New York State Agricultural Experiment Station.

1. Dick, G. F., and Dick, Gladys H.: The Bacteriology of Dried Powdered Milk Preparations Used in Infant Feeding, Am. J. Dis. Child. **34**:1040 (Dec.) 1927.

RESULTS

The following prepared infant foods were investigated: Dextri-Maltose (Mead Johnson & Company): Nineteen samples of Dextri-Maltose were examined, and no hemolytic or *viridans* type of streptococci was noted.

Lactic Acid Milk (Mead Johnson & Company): Fourteen samples of lactic acid milk (cultured) and ten of lactic acid milk (acidulated) were examined. No hemolytic or *viridans* streptococci were found. In some instances, the lactic acid milk which had been prepared by a lactic acid producing culture showed a large number of such organisms present. Such a condition meets the needs of certain pediatricians who require the product to be prepared in such a manner that such lactic acid producing types of organisms are present in the final product.

Powdered Milk (Mead Johnson & Company): Four samples of powdered half skim milk and three samples of powdered whole milk were examined, and no hemolytic or *viridans* type of streptococci was noted.

Boilable Protein Milk (Mead Johnson & Company): Eight samples of Boilable Protein Milk showed no evidences of the hemolytic or *viridans* type of streptococci. Many of these samples were practically free from any type of organism. Although boiling reduced the total number of organisms, the reduction was negligible, as a majority of the types were present in the spore stage. These spores are able to resist boiling temperatures for a short period.

Recolac (Mead Johnson & Company): Twelve samples of Recolac examined showed no indication that the hemolytic or *viridans* type of streptococci was present.

Casec (Mead Johnson & Company): The eighteen samples of Casec which were examined showed no indication of the hemolytic or *viridans* type of streptococci. Samples of Casec which had been boiled for two minutes previous to analysis showed a material reduction in the total number of organisms present.

Dryco (the Dry Milk Company): Only one sample of Dryco was examined, but no hemolytic or *viridans* type of streptococci was noted.

Similac (Moores & Ross, Inc.): Twenty-one samples of Similac weré examined, nine of which had been prepared by the process recently put into practice by this concern. In two of the samples prepared by the older process, a small number of the *viridans* type of streptococci was found, but in none of the samples were hemolytic streptococci encountered.

Karo Syrup (Corn Products Refining Company): Six samples of Karo Syrup were examined, two of which were the Red Label and the remainder of the Blue Label variety. One of these samples showed a

few colonies of the *viridans* type which, on subsequent study proved not to be streptococci.

Modillac (Wm. S. Merrell Company): Six samples of Modillac showed no indication that hemolytic or *viridans* streptococci were present.

Mellin's Food (Mellin's Food Company): No hemolytic or *viridans* type of streptococci was found in twelve samples of Mellin's Food which were secured and examined.

S. M. A. (Laboratory Products Company): Eight samples of S. M. A. were obtained, five of which were in powder form and three liquid. One of the powdered samples contained a small number of suspicious colonies of the *viridans* type, but no satisfactory isolations could be made.

Lactogen (Nestlé's Food Company): Three samples of Lactogen were analyzed, with no indication of the hemolytic or *viridans* type of streptococci.

Nestlé's Milk Food (Nestlé's Food Company): No hemolytic or *viridans* type of streptococci was found in three samples of Nestlé's milk foods.

COMMENT

Examination of a representative series of prepared infant foods revealed no appreciable number of hemolytic or *viridans* types of streptococci. No information is available at present to indicate whether this condition is brought about by efficient methods of manufacture or is due to the absence of this type of organism in the raw product. It is possible that the latter condition exists. Related experiments indicate that certain types of hemolytic streptococci which occur in a variety of milk and related products may survive the temperatures to which the prepared infant foods are submitted during their preparation. However, it has been found that a majority of the concerns now producing commercially prepared infant foods are cognizant of the fact that hemolytic streptococci are sometimes heat resistant and may appear in their products if necessary precautions are not adhered to during certain stages of the manufacture. In all of the samples of prepared infant food examined at the New York State Agricultural Experiment Station, there has been no indication that any brand of such food that has been subjected to routine inspection has been grossly contaminated with hemolytic streptococci.

Because of the probabilities that the raw products from which infant foods are manufactured may contain hemolytic streptococci as well as the possibility of contamination by such organisms during the manufacturing process, it would seem advisable, however, that a routine control be established by the concerns over their respective products or by some central agency. Such control need not be elaborate; it could be

carried out with a small expenditure and would insure the absence of the group of hemolytic streptococci which may or may not be significant from a standpoint of health. If subsequent investigations prove that the type of hemolytic streptococci which appears sporadically in dried milk products is nonpathogenic to infants, such routine control might be discontinued.

CONCLUSIONS

Examination of a representative series of samples of prepared infant foods revealed practically no hemolytic streptococci.

It is suggested, however, that until more information is available a medium containing blood, used as a routine control, might alleviate further suspicion and possibly bring to light any occasional infection of the infant food either during the manufacturing process or in the raw products.

THE ACIDOSIS OF ACUTE DIARRHEA IN INFANCY*

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Our purpose in the present work was to investigate the cause of acidosis in the condition which is variously called summer diarrhea, acute dehydration, acute intoxication, etc. The infants used in this study all presented the classic symptoms: diarrhea, vomiting, dehydration (as diagnosed by the sunken fontanel, sunken eyeballs and loss of elasticity in the skin) and a more or less marked prostration. No cases were included which did not clearly show these symptoms. Two of the patients were later found to be suffering from a dysenteric infection, but as neither the symptoms in the acute stage nor the chemical composition of the blood differed in any way from the rest of the group, these cases have been included in this report. The samples of blood were taken while the patient was fasting and before any treatment had been given.

It is not possible to obtain from dehydrated infants enough blood in one sample for a complete analysis of all the factors influencing the acid base relationships. We were prepared to proceed in stages. The work was begun by determining the following factors: total fixed base,[1] chlorine [2] and carbon dioxide combining power.[3] The last mentioned factor has been taken, not quite correctly, of course, to represent the degree of acidosis and the amount of base bound as bicarbonate. The total carbon dioxide content and the p_H of the blood would have given us accurate information as to these two factors (degree of acidosis and base bound as bicarbonate) but as we were not, in any case, going to draw conclusions from other than marked variations (the normal variations being considerable) it was thought that the carbon dioxide combining power would give us sufficiently accurate information for our purpose.

In obtaining figures for total base, chloride and bicarbonate, we obtain also, indirectly, a figure representing the sum of all other blood

* Submitted for publication, Feb. 16, 1929.

* From the Harriet Lane Home of the Johns Hopkins Hospital and the Department of Pediatrics of the Johns Hopkins University Medical School.

1. Fiske: J. Biol. Chem. **51**:55, 1922.
2. Fiske and Sokhey: J. Biol. Chem. **63**:310, 1925.
3. Van Slyke: J. Biol. Chem. **61**:523, 1924.

acids (phosphorus, sulphur, organic acids and protein). This value which, of course, is obtained by subtracting from the total fixed base the sum of chloride and bicarbonate, we have called the undetermined acid. As both the method for fixed base and the method for chloride determinations give extremely accurate results, the accuracy of the value for undetermined acids is determined by the accuracy of the bicarbonate values.

TABLE 1.—*Changes in Base, Chloride and Bicarbonate in Acute Diarrhea*

Substance	Normal Range, Cc., Tenth Normal	Number of Cases		
		Normal	Low	High
Total base	154-161	5	14	6
Chloride	95-108	7	10	8
Bicarbonate	19- 27*	13	11	1
Undetermined acid	25- 35	12	1	12

* This corresponds to from 40 to 60 per cent by volume. We have put the lower limit as low as 40 per cent by volume in order to be sure to include in the acidosis group only patients with unquestionable and rather marked acidosis.

TABLE 2.—*The Acid-Base Balance in the Serum in Acute Diarrhea*

Case	Bicarbonate, Cc., Tenth Normal	Chloride, Cc., Tenth Normal	Undetermined Acid, Cc., Tenth Normal	Total Base	Base Bound as Chloride, per Cent
2	26	70	48	145	48
3	19	85	55	158	54
4	10	132	30	172	77
5	20	94	41	155	61
6	25	82	43	150	55
7	8	134	43	185	72
8	8	139	32	179	78
9	9	112	33	154	73
10	6	131	25	162	81
11	21	121	32	174	70
12	19	97	31	147	66
13	16	100	36	152	66
14	14	95	31	140	68
16	12	115	31	158	73
17	18	90	43	151	60
18	19	89	40	148	60
19	15	102	35	152	67
20	24	84	41	149	56
21	20	97	45	162	60
22	21	83	35	139	60
23	28	71	46	145	49
24	11	95	32	138	69
25	23	81	36	140	58
26	25	98	34	157	62
27	15	114	22	151	76

It may be seen in table 1 that the most common change was a lowering of the total fixed base of the serum. This decrease of base was often considerable (table 2). After the base, the chloride content was most often found abnormal, and the aberrations from the normal were about equally common in either direction. The bicarbonate values were about equally divided between normal [4] and low values; the values for

4. As mentioned before, the normal limits were taken as ranging from 40 to 60 per cent by volume, and among the "normal" values a few are included, therefore, which more appropriately, perhaps, should have been labeled as mild acidosis (table 2).

undetermined acid were about equally divided between normal and high values.

One of the two acid factors, chloride and undetermined acids, must have been responsible for the acidosis in those cases in which this condition was present. When investigating which of these two factors is responsible, one should remember that the amount of acids present in the blood must be exactly the same as the amount of base. If, of two infants, one has both higher base and higher chloride concentration in the serum than the other, it seems possible that the higher concentration of chloride is due to the fact that there is more base there to bind. If, on the other hand, of two infants with the same base concentration in the serum, one has a higher concentration of chloride, this fact must be due to some other factor than the base. In investigating the difference in acid concentration one should, therefore, as far as possible, compare only bloods with about the same amounts of base.

This precaution has been taken in chart 1 in which the cases have been arranged according to the amount of fixed base in the serum. Each dot on the curves represents one case, and at the foot of the figure may be seen which case is indicated. The case placed farthest to the left is the one in which the total fixed base was lowest; the case farthest to the right, the one in which the total fixed base was highest. If the values for fixed base were plotted as a curve in the diagram, this curve would show, therefore, a gradual rise from left to right. Consequently, two adjacent cases nearly always have about the same amount of fixed base.

It is obvious that when arranged in curves this way, the acid factor responsible for the acidosis will betray itself by moving in a direction opposite to that of the bicarbonate. A low bicarbonate value has been taken as being synonymous with acidosis; if, of two infants with about the same amount of base, one has a normal, the other a low, bicarbonate value, it is clear that the acid responsible for the low bicarbonate must be higher in the second case than in the first. In chart 1 may be seen how the curve representing the chloride values is practically an inversion of the curve representing the bicarbonate values. When the bicarbonate value goes up, the chloride value goes down, and vice versa. It is, consequently, the hydrochloric acid that is responsible for the acidosis present. As for the values for undetermined acids, the curve representing these values is often parallel to the curve representing the bicarbonate values. This fact excludes the possibility that any of the acids included in the values for undetermined acids (phosphate, sulphate organic acids, protein) might to any appreciable degree be responsible for the acidosis in acute intoxication.

The fact that the acidosis in acute dehydration is a chloride acidosis is not new; it was previously described by Schiff [5] and by Hartmann.[6] We want to point out, however, that a high content of chloride in the blood is not necessary to the development of chloride acidosis. The condition may also be due to a low value for base (cases 13, 14, 19 and 24). It is the relationship between base and chloride that is of importance,

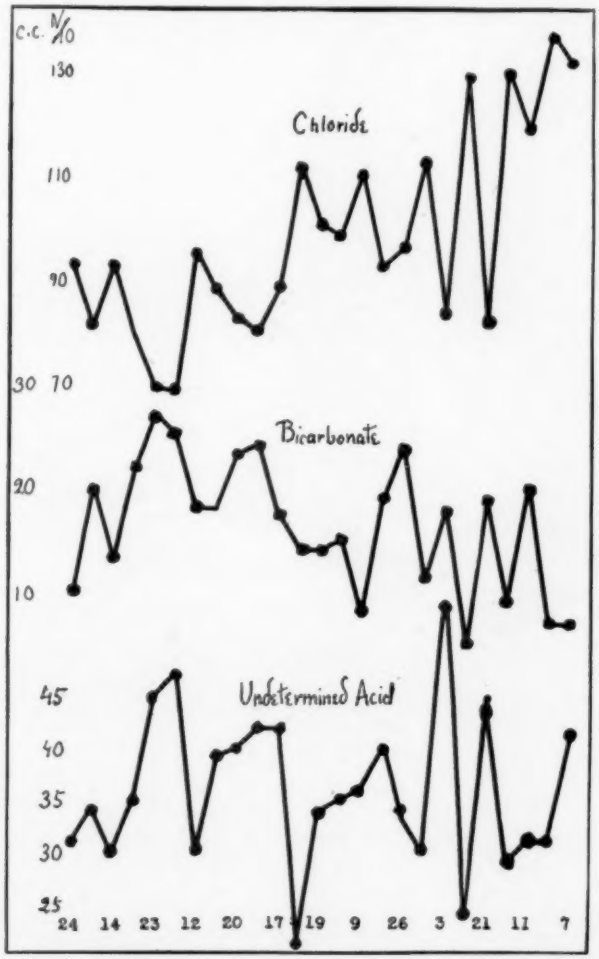

Chart 1.—Cases ranged according to the amount of fixed base. The curve for chloride and the curve for bicarbonate move in opposite directions; the curve for undetermined acids shows some degree of parallelism to the curve for bicarbonate. Consequently, chloride, and not undetermined acids, is responsible for the acidosis, when present.

5. Schiff, Bayer and Fukuyama: Jahrb. f. Kinderh. **119**:161, 1928.

6. Hartmann, A. F.: Chemical Changes Occurring in the Body as a Result of Certain Diseases, Am. J. Dis. Child. **35**:557 (April) 1928.

and when the chloride increases proportionately more than the base, or decreases proportionately less, a chloride acidosis results.

The logical way to demonstrate the truth of this statement is to express the base bound as chloride in percentage of the total base. In chart 2, the middle curve represents the values so obtained, and the cases are ranged according to the size of these values. Farthest to the

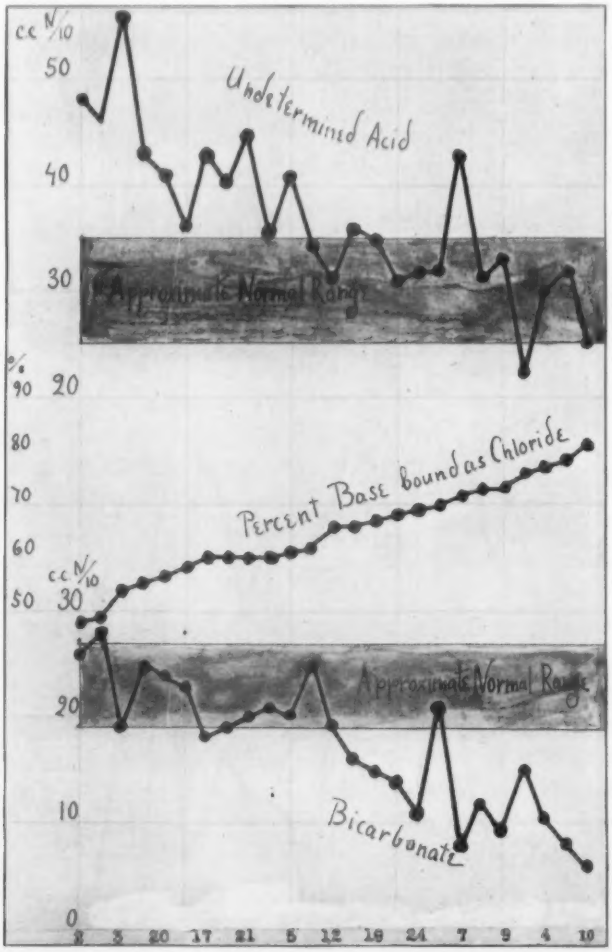

Chart 2.—Cases ranged according to the amount of base bound as chloride. When the percentage of base bound as chloride is low, the place of the chloride is taken up by undetermined acids. When the percentage of base bound as chloride is high, the chloride takes the place of the bicarbonate.

left, consequently, are those cases in which an abnormally small percentage of the total base was bound as chloride; farthest to the right, are those cases in which a high percentage of the base was so bound. The lower curve gives the bicarbonate values, and it may be seen that

the slope of this curve is opposite to that of the curve representing the percentage of base bound as chloride. In other words, the cases of acidosis are those in which a high percentage of the base is bound as chloride.

The diagram also throws further light on the behavior of the undetermined acids. The upper curve in the diagram represents undetermined acids. High values for undetermined acids are to be found in the cases in which the chloride content is abnormally low. To the left are low chloride values, high values for undetermined acids and normal bicarbonate values. Toward the middle of the diagram can be seen

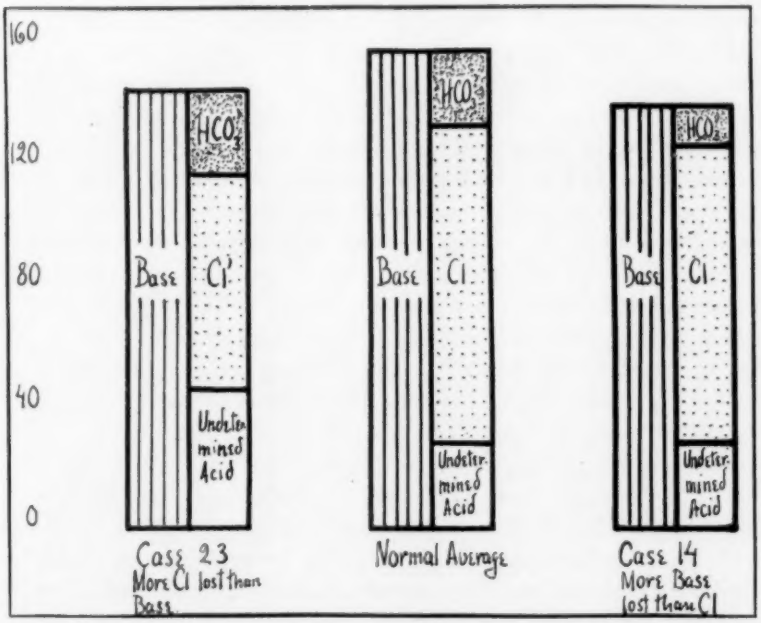

Chart 3.—The proportion of values for base and chloride in two cases, compared with the normal values. When there is too little chloride in the blood, an adjustment is made by the undetermined acids. When there is too much chloride, an adjustment is made by the bicarbonate.

how, with increasing chlorides, the undetermined acids drop down into the normal range and remain there, while the bicarbonate values begin to drop down below the normal range (chart 3).

Chart 2, consequently, gives an interesting picture of how the body meets changes in blood chloride in these cases. When the chloride content is high, it pushes bicarbonate out of the blood; when it is low, its place is filled by undetermined acids. The question as to which of the undetermined acids fills this function is partly answered by the fact that the difference between the lowest and the highest value for undetermined

acids in our cases was 33 cc. tenth normal. None of the undetermined acids alone can have been responsible for such variations; several or all of them must have contributed.

It was said that when chloride content is low its place is filled by undetermined acids. This is not quite true, as a low chloride value nearly always is associated with a low base value. Gamble and Ross [7] showed that losses of chloride due to vomiting are met in three ways: decrease of base, increase of bicarbonate and increase of undetermined acids. In our cases only two of the three phenomena were present. In no case was there an alkalosis. It might be possible that this way of reacting to loss of chloride is peculiar to infants, but to judge from determinations made in three patients with pyloric stenosis (bicarbonate 30, 35, and 52 millimols) and one patient with habitual vomiting (bicarbonate 35 millimols), alkalosis may develop in a vomiting infant just as in a vomiting adult. It may therefore be supposed that there is something inherent in the condition of acute intoxication which prevents the development of alkalosis even in the cases in which a low chloride content seems to invite the bicarbonate to increase. This same tendency is probably responsible for the fact that even normal bicarbonate values were practically never found unless there was some decrease of the chloride below normal (cases 2, 6, 20, 23, 25 and 26).

It was stated that the acidosis of acute intoxication seems in most cases to be due to abnormal proportions between chloride and fixed base. The manner in which this disproportion develops is probably different in different cases. It seems to us that two distinct types of chloride acidosis are apparent: the type with high base in which the chloride is increased even more than the base, and the type with low base in which the chloride is not decreased in proportion to the base (chart 4). The latter type might probably be explained by losses from the stomach and the intestines, losses which are not replaced because of the vomiting and the unsatisfactory absorption.

The stools are much richer in base than in chloride. Holt, Courtney and Fales [8] found in six patients with severe diarrhea an average base secretion of 648 cc. daily and an excretion of chlorine of 80 cc. tenth normal daily. On the other hand, the fluid lost by vomiting may contain free hydrochloric acid, that is, more chlorine than base, although in the children here in question one would have suspected perhaps a rather low acidity of the gastric content (Marriott and Davidson,[9]

7. Gamble and Ross: J. Clin. Investigation **1**:403, 1924.

8. Holt, L. E.; Courtney, A. M., and Fales, H. L.: The Chemical Composition of Diarrheal as Compared with Normal Stools in Infants, Am. J. Dis. Child. **9**:213 (March) 1915.

9. Marriott, W. M., and Davidson, L. T.: Acidity of Gastric Contents of Infants, Am. J. Dis. Child. **26**:542 (Dec.) 1923.

Ylppö [10]). To judge from our series of observations there is about an equal chance whether an infant with intoxication will lose more base than chloride (and develop acidosis) or more chloride than base. We do not consider it correct to state that the acidosis is due to loss of base bound as bicarbonate (Hartmann [6]). Loss of base bound to any acid other than hydrochloric acid (phosphate, sulphate, organic acids or protein) would result in a chloride acidosis, provided the loss were not balanced by the loss of an equivalent amount of hydrochloric acid from the stomach. Loss of base bound as bicarbonate, on the other hand,

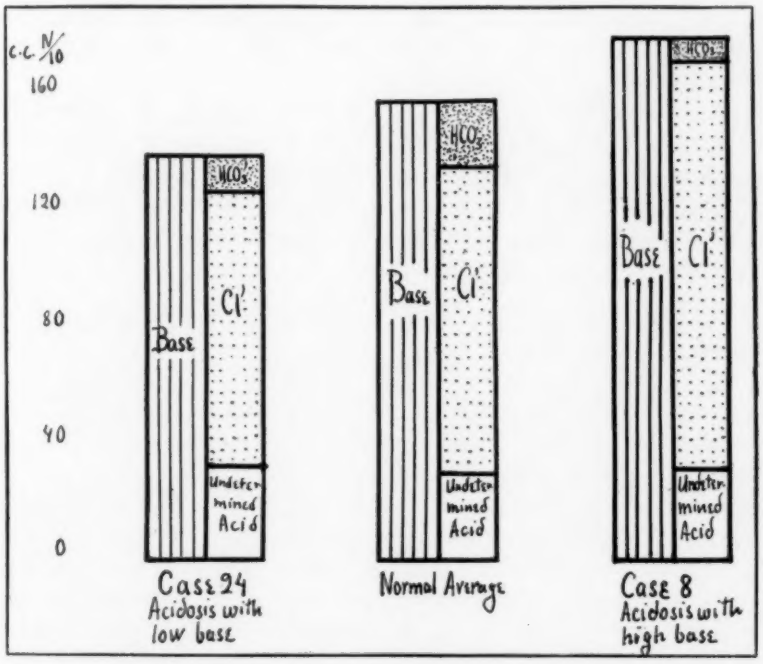

Chart 4.—The proportion of values in two types of acidosis found in intoxication.

would not result in a chloride acidosis if an equivalent amount of hydrochloric acid were lost from the stomach. The chloride acidosis is due to the loss of base not bound as chloride and not compensated for by a loss of hydrochloric acid.

Of the second type of acidosis with high base, we have only a few cases. We know from the pioneer work of Schiff [5] on experimental dehydration that this type of acidosis may occur in pure dehydration without diarrhea. This fact does not, of course, exclude the possibility

10. Ylppö: Acta paediat. **3**:213, 1923.

that in the pathogenesis of this type of acidosis in our cases diarrhea may have played a rôle, but, whether or not that is so, other factors must also be involved. These factors are probably blood concentration [11] and functional changes in the kidney. It is impossible at present to evaluate the influence of these factors on the composition of the blood; we hope to throw some light on this question by balance experiments and determinations of blood volume in cases of acute intoxication.

SUMMARY AND CONCLUSIONS

In a series of twenty-five cases of acute intoxication, severe acidosis was found in eleven. The most common change in the blood was a decrease in the concentration of fixed base, which occurred in fourteen cases.

The acidosis, when present, was a chloride acidosis. There were two types: one with low base, chloride not being decreased in proportion to the base; and one with high base, chloride being increased even more than the base. The cause of the acidosis is probably loss, in the stools, of base not bound as chloride and not compensated for by loss of hydrochloric acid through vomiting.

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11. Buckmann, T. E., and Darrow, D. C.: Volume of Blood, Am. J. Dis. Child. **36**:248 (Aug.) 1928.

THE TREATMENT OF JUVENILE DIABETES*

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The physician who treats diabetic children today aims to have them reach adult life with as good physical development as possible. They should be able to carry on their daily life like normal children of their own age.

Before the discovery of insulin and for a period afterward, many authors stressed the importance of keeping diabetic children entirely sugar-free. While one still attempts to keep them sugar-free, one may in so doing defeat the primary purpose, namely, the production of normal growth and development. In other words, in attempting to keep the urine entirely sugar-free one may be forced to prescribe a low caloric diet which is not compatible with normal gains in weight and height. By increasing the diet and dosage of insulin, a child may develop glycosuria some time during the twenty-four hours, yet, in spite of the glycosuria, gain in height and weight. Thus the presence of an occasional or low-grade glycosuria may be compatible with normal growth and development.

GENERAL MANAGEMENT

There are twenty diabetic children under constant observation at the Mount Sinai Hospital outpatient department. In nearly every instance they are entirely reliable as regards their intake of food and dosage of insulin. The new diabetic child is preferably placed in the wards of the hospital for a period of time so as not only to study his tolerance and the amount of insulin he will require, but also to educate and train him in the new mode of life which he will have to follow. After a time the mothers and even the grandmothers of the children are invited to visit the diet kitchen to learn the methods of preparation of the food that the child will eat when he returns to his home.

All children are required to keep a record of all food eaten; some keep a daily record; others, as a reward for previous accuracy, bring just a list of all the foods eaten on the day previous to coming to the clinic. A specimen of each urine voided during the previous twenty-four hours is brought to the clinic and examined. If there is any doubt as to the substitution of urines, a specimen is collected in the clinic. It has not been found necessary to make routine estimations of the blood sugar. More information is obtained from urinalyses of individual

* Submitted for publication, March 29, 1929.
* From the Department of Pediatrics, Mount Sinai Hospital.
* Read before the Harlem Medical Society, Feb. 26, 1929.

specimens than from a single test of the blood sugar, especially when the morning dose of insulin has had to be postponed in order to determine in the clinic the blood sugar during fasting.

Administration of insulin has in every instance been thoroughly supervised, and to guard against possible emergencies, at least two members of the patient's family besides himself have had to demonstrate their ability to inject insulin. All children over 9 or 10 years of age inject insulin into themselves. A social worker visits the homes of the children at a meal hour at stated intervals, always unannounced, in order to see whether or not the instructions are being properly carried out. Her reports are uniformly good even in the poorest homes. Each child visits the clinic every two to three weeks, and broken appointments are accounted for immediately. This is an important point in the organization, as a regular follow-up goes a long way toward preventing laxness on the part of the patient or the family. During the past two years, only two children of the group have entered the hospital in a state of coma; one child, who previously had been in coma, admitted a serious break in her diet; the second child, in spite of adequate treatment, went into coma during an attack of hay-fever and bronchial asthma.

DIETARY MANAGEMENT

Opinions vary as to the amount of carbohydrate, protein and fat that a diabetic child should receive, although it is generally agreed that it is well to keep the children slightly under weight. Even with a restricted diet, there seems to be a marked tendency to retention of water when insulin is used, and gains in weight occur rapidly. The diet high in carbohydrates advocated by Sansum,[1] and Geyelin and Mackie,[2] and others in this country and by Adlersberg and Porges [3] in Vienna has revolutionized the dietary management in many cases. Allen [4] and Joslin [5] now treat their children with diets higher in carbohydrates than formerly; yet they do not go to the extreme of 300 Gm. of carbohydrate a day as advocated by Sansum. It is difficult to understand how a diabetic organism can burn nearly as much carbohydrate as a normal one even with insulin, but it is true that a moderate amount of carbohydrate is well tolerated by diabetic children. On the other hand,

1. Sansum, W. D.; Blatherwick, N. R., and Bowden, R.: The Use of High Carbohydrate Diets in the Treatment of Diabetes Mellitus, J. A. M. A. **86**:178 (Jan. 16) 1926.

2. Geyelin, H. R., and Mackie, T. T.: Diabetes Mellitus in Children, N. Y. State J. Med. **29**:677 (June 1) 1929.

3. Adlersberg, D., and Porges, O.: Zur Therapie und Praxis der kurativen Diabetesbehandlung, Klin. Wchnschr. **5**:1451, 1926.

4. Allen, F. M.: Present Results and Outlook of Diabetic Treatment, Ann. Int. Med. **2**:203, 1928.

5. Joslin, E.: Arteriosclerosis and Diabetes, Ann. Clin. Med. **5**:1061, 1927.

it is dangerous in outpatient work to experiment with a high carbohydrate intake, and the average child therefore gets from 80 to 100 Gm. of carbohydrate a day. The amount, of course, varies with the age of the child. Children under 4 years of age receive 75 Gm. of carbohydrate a day; those from 4 to 7 years of age receive from 80 to 90 Gm., and those from 10 to 14 years, from 100 to 130 Gm. daily. These figures are exclusive of the amount of calculated carbohydrate in protein and fat. The amount of carbohydrate is usually divided so that about two fifths of the daily amount is given at breakfast and at supper and one fifth at lunch. For smaller children, or in those who have reactions to insulin, the diet is subdivided into five meals a day; a small amount of carbohydrate, usually from 5 to 10 Gm., is given at 10: 30 a. m., or from about three to three and a half hours after the morning dose of insulin, and again at 9: 30 p. m., three and a half hours after the evening dose. Severe reactions to insulin can for the most part be avoided in this way.

Diets high in protein are not advocated by any one so far as I know. The general trend is to give enough protein to assure a positive nitrogen balance. An excessive amount of protein elevates metabolism and may cause acetonuria.[6] The average protein content of the diets is from 55 to 60 Gm. (from 1.5 to 3 Gm. per kilogram) per day or from 10 to 15 per cent of the total calories.

Priesel and Wagner [7] advocated a diet high in fat; from 125 to 200 Gm. a day. This amount, however, constantly produces acetonuria. The older children therefore receive from 90 to 120 Gm. of fat daily and the younger ones from 50 to 80 Gm., the carbohydrate-fat ratio usually averaging 1: 1. Lowering the intake of fat reduces the caloric intake somewhat, but I have as yet seen no harm from this reduction. Wagner reported adequate growth and development in his children on diets high in fat and did not place any emphasis on the presence of acetone. Most authors, however, feel that a diet high in fat raises the insulin requirement over a certain period, while a diet containing a moderate amount of carbohydrate and fat, such as outlined in the foregoing, does not. Furthermore, a diet high in fat raises the body weight, which, according to Joslin, increases the severity of the diabetes. Joslin is also somewhat concerned over the premature development of atherosclerosis in diabetic patients, and he attributes its appearance, at least in part, to the high fat content of the diet with which many of his patients formerly were treated. Roentgenograms have been taken of all of the diabetic children at Mount Sinai Hospital, and as yet atherosclerosis has not been noted.

6. Widnäs, K.: Diabetes Mellitus in the Child, Acta pediat. **8**:1 (Sept.) 1928.

7. Priesel, R., and Wagner, R.: Die Pathologie und Therapie der kindlichen Zuckerkrankheit, Ergebn. d. inn. Med. u. Kinderh. **30**:536, 1926.

Treatment for infections consists in reducing the total caloric intake, especially the protein and fat content, and, in order to avoid acidosis, maintaining or even increasing the amount of carbohydrate. At the same time, the dosage of insulin is increased, frequently both in the total number of units and in the number of daily injections, until the infection has subsided and the lost tolerance has begun to return. The treatment for coma is too lengthy a subject to discuss in this paper.

INSULIN

All of the diabetic children in the Mount Sinai Hospital now receive insulin. During the past two years five children were treated without it. They did well for a time. Subsequently they lost their tolerance, became sugar-free with difficulty on a somewhat lower caloric intake and did not gain weight or grow consistently. At the onset of diabetes one frequently sees this apparent increase in tolerance with a restricted diet, and if insulin is being given, it may be discontinued. The sequence of events in such a case is that for some time, usually from three to nine months, a child will remain sugar-free without insulin; then, generally as the result of a slight infection, glycosuria recurs. It will now be found that the glycosuria is not so readily controlled as previously and that insulin will be required to keep the child sugar-free. The administration of synthalin according to the method advocated by Frank and his followers at the Breslau Clinic was tried in some cases. This drug caused loss of appetite and general malaise and was therefore discontinued after a fair trial.

Fifteen of the children receive two injections a day, the maximum dose being 35 units. Five children receive three injections a day with a maximum of 62 units. Subdividing the dosage of insulin when it seems that there is a loss of tolerance is frequently effective. One of the children now receiving three injections a day formerly received 85 units divided into six injections a day; that is, one injection every four hours. Under this regimen she recovered part of her tolerance, so that the number of injections was reduced to five and later to four a day. Last summer while the child was at a summer camp the number was further cut down to three injections a day. Her diet remains unchanged, and since last summer she has been sugar-free on 54 units of insulin daily, subdivided into three injections. Several children who have been receiving two daily injections of insulin have had an additional injection for a short time when there was a loss of tolerance with glycosuria. Usually improvement is noted, tolerance returns and the additional injection is discontinued. None of the children gets one injection a day. I feel that insulin is required more than once in twenty-four hours if there is a true diabetes, the endogenous insulin not being sufficient to carry the child along with only

one therapeutic dose a day. Reactions to insulin are frequent, usually mild and readily controlled; occasionally they are severe. Fortunately, they can be successfully combated by giving a small amount of carbohydrate by mouth. Insulin shocks rarely are severe enough to warrant an intravenous injection of dextrose. Usually the child recovers without treatment.

Space does not permit a discussion of the prognosis of diabetic children. Their general health and appearance are excellent. The majority are about 10 per cent under the normal height for their age, but their weight is in most instances normal or slightly above normal for their height. There seems to be a delay in reaching puberty in children who have had diabetes, at least from the ninth or tenth year of life. Definite statements, however, cannot be made until the youngest patients who are being treated reach puberty. The hospital keeps a regular photographic record of all the patients, and their physical development can best be observed in this way.

CONCLUSIONS

1. While restrictions of diet and substitution therapy in the form of insulin are cumbersome, they constitute the only means by which diabetic children can be brought to adult life.

2. There are different standards for calculation of the diet in different clinics. More statistics will have to be collected in order to determine the optimum method, but the results in most clinics are generally satisfactory.

3. There is, however, sufficient evidence to justify a more liberal carbohydrate diet than formerly advocated.

Case Reports

CHRONIC TETANY AND CHRONIC MYCELIAL STOMATITIS IN A CHILD AGED FOUR AND ONE-HALF YEARS *

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In the past twenty years, a vast amount of work has been done which has broadened our views concerning tetany. Recently, this extensive literature was reviewed and correlated by MacCallum,[1] himself one of the foremost contributors. He pointed out that "there may be several types of tetany differing widely in their etiology and in the mechanism of their production although the final changes in the blood which bring about the actual symptoms may be the same." He agreed with Aschenheim that those cases of tetany that are related to the psychoses, infections, intoxications and endocrine disorders can be classified only tentatively, but he demonstrated at least four well studied types: (1) that resulting from extirpation of the parathyroid glands, (2) gastric tetany, (3) tetany from forced respiration and (4) infantile tetany or spasmophilia. He also pointed out the possibility that other types are produced by modification of the diet and by artificial disturbance of the constituents of the blood through administration of huge doses of various salts.

The case reported here represents a chronic form of tetany which suggests the presence of definite parathyroid deficiency. The hypocalcemia was associated with a chronic intractable mycelial infection of the mouth. Both conditions are sufficiently rare to warrant this presentation.

REPORT OF CASE

History.—E. P., a girl, aged 4¼ years, was first seen by one of us on June 28, 1926, in the pediatric dispensary of the hospital of the University of Pennsylvania, and was admitted to the ward for study. The chief complaint was frequent convulsions.

* Submitted for publication, Feb. 5, 1929.

* From the Department of Pediatrics of the University of Pennsylvania, the University Hospital, and the William Pepper Laboratory of Clinical Medicine.

1. MacCallum, W. B.: On the Pathogenesis of Tetany, Medicine **3**:137 (May) 1924.

She was born Oct. 1, 1921, after normal labor, and was breast fed for nineteen months. Solid food was introduced into the diet at one year, and her usual dietary regimen seemed to have been fairly adequate. She had never received cod liver oil or regular exposure to ultraviolet light. She was reported as having sat up at 6 months, walked at 7 months and talked at 15 months. She was always rather quiet and unsociable and was not active. Her parents and two other living children were normal. The mother had had no miscarriages, and no familial history of chronic disease was reported. The patient was said to have had "summer diarrhea" in 1922 and 1923, pneumonia in 1924 and 1925, measles, German measles, frequent boils and infections of the upper part of the respiratory tract. Tonsillectomy and adenoidectomy had been performed in November, 1925.

The child had had an attack of dyspnea and convulsions shortly after the tonsillectomy. The infection of the mouth had appeared about the same time. She had never fully recovered, and was subject to frequently recurring attacks of convulsions or "muscular spasms and twitchings." In January, 1926, she was given a course of twelve intravenous injections of calcium lactate by a local pediatrician. This treatment brought about an improvement for two months,; then the symptoms returned slowly, and steadily grew worse. These spasms were not influenced by fever or other symptoms, and, despite the recurrent attacks, her health remained fairly good, and she continued to grow. Except for painful micturition, no other symptoms were noted. Her mentality and cooperation seemed normal to her parents.

Physical Examination.—The patient was 39 in. (99.5 cm.) tall and weighed 34 pounds (15.4 Kg.); the temperature and the pulse and respiratory rates were normal. She appeared slightly obese but well developed, and exhibited a rather sluggish disposition. Her skin was dry and rough, but there was no evidence of myxedema. The positive observations were: carpopedal spasm; positive Chvostek and Trousseau signs; a diffuse stomatitis resembling thrush; small, soft, ridged teeth with moderate pyorrhea; slight pallor of the mucous membranes; moderate cervical adenitis; ridging and distinct softening of finger and toe nails, and evidences of moderate healed rickets. The hair was dry, but not coarse; enlargement of the thyroid or thymus glands could not be demonstrated; the liver and the spleen were not palpable, and there was no distinct change in the genitalia. Aside from these, there were no noteworthy observations.

Laboratory Observations.—The blood count was normal. The blood calcium (determined by the method of Collip, a modification of the Kramer-Tisdall method[2]) was 4.8 mg. per hundred cubic centimeters; the blood phosphorus (determined by the method of Fisher and Subbarow[3]) was 11.4 mg. per hundred cubic centimeters. The urine showed many pus cells, and there was an elimination of 65 per cent of phenolsulphonphthalein in two hours. The feces did not show any ova or parasites. The Wassermann reaction of the blood was negative, the blood carbon dioxide was 52 per cent by volume and the blood urea nitrogen was 15 mg. per hundred cubic centimeters.

Treatment and Course of Illness.—On admission to the hospital, the patient was given calcium chloride, 20 grains (1.3 Gm.) every three hours over a period of five days, and this was supplemented by a well balanced diet, which included 32 ounces (946 cc.) of milk and 1 ounce (32 cc.) of cod liver oil per day. She also received exposures to the "Alpine" lamp. At the end of this time, she was much improved, had lost the carpopedal spasm and Trousseau's sign, but still

2. Collip, J. B.: J. Biol. Chem. **63**:461 (March) 1925.
3. Fisher and Subbarow: J. Biol. Chem. **66**:375 (Dec.) 1925.

showed the Chvostek sign and the electrical reactions typical of tetany. The blood calcium was reported as being 9 mg. per hundred cubic centimeters. Her regimen remained the same until the time of her discharge, except that the calcium dosage was cut to 60 grains (3.88 Gm.) per day. She was discharged on Sept. 3, 1926, owing to the fact that she had developed gonorrheal vaginitis. During the two months of her stay, it was seen that a high calcium level could not be maintained by the therapeutic measures used, none of the subsequent determinations reported being above 8 mg. per hundred cubic centimeters.

On September 6, she was readmitted to the isolation department because of a recurrence of the convulsions and carpopedal spasm. The blood calcium had dropped in a week from 7.9 to 5.2 mg. per hundred cubic centimeters. She was given 30 grains (1.9 Gm.) of calcium chloride by mouth every three hours for eight doses. The carpopedal spasm disappeared in six hours and the Trousseau sign in fourteen hours. She was held under strict isolation until Sept. 20, 1926, but was given the treatment for tetany used during her previous stay in the hospital, supplemented by mercurochrome irrigations of the vagina. Unfortunately, no determinations of the serum calcium were made at this time.

From Sept. 24, 1926, until July, 1927, she was under observation in the dispensary. During this period, the vaginitis was cured, this treatment being carried out in the gynecologic dispensary.

Therapy at the pediatric dispensary was directed toward maintaining her free from active tetany and toward clearing up the oral infection. The results along both lines were unsuccessful, despite the fact that many procedures were given adequate trial.

In the main, we depended on a balanced diet, large doses of calcium and cod liver oil and ultraviolet light. During periods of marked increase of the tetany, she was given 20 units of Collip's parathyroid extract daily until active symptoms disappeared. It was clearly observed that measures ordinarily successful were incapable of maintaining the normal or a high calcium level in this patient. For a period of three months, powdered parathyroid extract, 0.3 Gm.,[4] and thyroid extract, 0.12 Gm. daily were given. No improvement was noted.

All procedures tried for the conditions in the mouth proved equally unavailing. The following methods were used: 1 per cent solution of gentian violet, 2 per cent solution of mercurochrome-220 soluble, 10 per cent arsphenamine in glycerin, a paste of sodium perborate, as advised by Bloodgood,[5] and finally a course of eight intravenous injections of arsphenamine, with the administration of sodium iodide by mouth in adequate dosage. Not the least degree of permanent improvement was noted.

During this time, the patient's appetite was good and her general nutrition seemed normal. Her weight increased from 36 pounds (16.3 Kg.) to 44 pounds (20 Kg.) in eight months. In June, 1927, a slight opacity of both corneas with increased lacrimation was noted.

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4. The parathyroid extract used was Parathor-mone (Lilly) of which 1 cc. equals 20 units. A unit is defined by Collip as "one one-hundredth of that amount of extract which produces on an average a 5 mg. rise in blood serum calcium in 20-Kilo dogs, over fifteen hours."

5. Bloodgood, J. C.: Oral Lesions Due to Vincents' Angina; What Every Physician Should Know About Its Recognition and Treatment, J. A. M. A. **88:** 1142 (April 9) 1927.

On July 11, 1927, she was brought to the dispensary after an absence of a month. She was ill with bronchitis and was readmitted to the ward.

In addition to the observations already recorded, the following facts were noted: temperature 102 F. pulse 140, respiration 60, acute rhinopharyngitis and bronchitis, photophobia, diffuse corneal ulceration and signs of active tetany, including positive Chvostek and Trousseau signs and carpopedal spasm. The blood calcium was 5.8 mg. per hundred cubic centimeters; the phosphorus was 10 mg. per hundred cubic centimeters. The electrical reactions were reversed, and the cathodal opening contraction gave a response at 3 ma. The blood count showed red cells, 3,240,000; hemoglobin, 65 per cent (Sahli); white cells, 19,400 with an appropriate increase in the percentage of polymorphonuclears. The urine was normal and there was no evidence of gonococci in a slight vaginal discharge; later, the mycelial organism was isolated from this discharge.

The patient was again treated with large doses of calcium chloride. Six days after admission, she was free from fever, the lungs were clear, the carpopedal spasm had disappeared, and she seemed better. She still showed an active Chvostek sign, and the blood calcium was 5.7 mg. and the phosphorus 10 mg. per hundred cubic centimeters. A roentgenogram of her chest showed that it was normal and there was no evidence of an enlarged thymus. The blood count again disclosed nothing abnormal except slight secondary anemia.

Roentgenograms of the head, pituitary fossa and long bones were reported negative. The Wassermann reaction of the blood was negative, the van den Bergh reaction was normal, the blood carbon dioxide was 52 per cent by volume, and intradermal injections of old tuberculin up to 1 mg. gave no reactions. A sugar tolerance test was done with the following results: The patient was given 84 Gm. of dextrose in 180 cc. of water, the fasting blood sugar being 113 mg. Specimens taken at thirty minutes, sixty minutes and 120 minutes were reported as showing 174, 252 and 283 mg. sugar per hundred cubic centimeters, respectively. The following morning, the blood sugar before breakfast was 108 mg., and the urine was free from sugar. We have no explanation for this abnormal sugar curve. The patient never showed glycosuria or other symptoms leading us to suspect diabetes.

Another remarkable feature of this case was the chronicity of the stomatitis. The lesions occurred on the lips, buccal mucous membrane, tongue, palate and pharyngeal mucous membrane, extending up to the nasopharynx and down the esophagus. These lesions consisted of a white, soft exudate which, when removed, left a bleeding ulceration. Over a period of a year, all of the various therapeutic measures mentioned before were thoroughly tried again, without success. In addition, (1) sodium iodide was given by mouth, up to 75 grains (4.8 Gm.) daily; (2) a concentrated solution of thymol in oil[6] was applied locally twice daily, and thymol, 1 grain (0.06 Gm.) in capsules was given; (3) compound solution of iodine was applied locally and (4) a 1 per cent solution of tartar emetic was given intravenously, the doses ranging from 0.5 to 4 cc. per day over a period of three weeks. Shelmire[7] recently advised the use of iodides intravenously in various fungous infections and advocated a solution composed of iodine 5 Gm., sodium iodide 10 Gm. and water 100 cc. Each cubic centimeter given was diluted to 25 cc. with distilled water. The first dose was 0.3 cc., and

6. Myers, H. B.: Unappreciated Fungicidal Action of Certain Volatile Oils, J. A. M. A. **89**:1834 (Nov. 26) 1927.

7. Shelmire, B.: Intravenous Iodine Therapy, Texas M. J. **22**:644 (Feb.) 1927.

this was gradually increased to 5 cc. of the stock solution. The only result obtained was thrombosis of the veins used. Finally the tongue and oral mucous membranes were painted with 0.5 per cent procaine hydrochloride, and 24 per cent trichloracetic acid was applied. This was repeated three times. During the period of desquamation following this rather strenuous treatment, a solution of gentian violet was used as a prophylactic, but the infection recurred promptly. The lesions extended up into the pharynx and down the esophagus, so that local applications did not have permanent effect. In addition, the infection was implanted by contamination in the vagina.

At first, smears and cultures of the lesions in the mouth showed the usual bacteria found there, plus the spirochete of Vincent and a yeastlike form thought to be *Monilia* or *Oïdium albicans*. This led to the use of local applications of ordinary yeast paste with the hope that the former would be outgrown by a nonpathogenic form. This was not successful.

Additional smears and cultures showed the presence of two organisms; a yeast, and a form with separate branching hyphae. On plain agar plates and Sabouraud's medium the hyphae only developed. The exact identity of the form has not yet been determined, but it closely resembles *Endomyces viullemini*.

COMMENT

MacCallum and Voegtlin [8] were the first to show conclusively that tetany experimentally produced in animals is characterized by low value of serum calcium, and that the active symptoms are relieved by the administration of calcium salts intravenously and by mouth. These observations, which have been confirmed by many, are almost universally accepted. Salvesen [9] showed that parathyroidectomized dogs can be kept alive for several years on a diet of milk and preparations rich in calcium. Luckhardt and Goldberg [10] showed that similar results can be obtained even on a diet of meat, if the administration of calcium is pushed far enough. Many observers have noted that although parathyroidectomized animals may be kept free from symptoms, the calcium of the serum cannot be forced to a normal level or maintained at a high level by the administration of calcium. Howland and Marriott [11] were the first to point out that the level of the serum calcium in infantile tetany, or spasmophilia is low, and that this condition can be relieved by the exhibition of calcium salts.

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8. MacCallum, W. B., and Voegtlin, C.: On the Relation of the Parathyroid Gland to Calcium Metabolism and to Tetany, J. Exper. Med. **11**:118, 1909.

9. Salvesen: Function of the Parathyroids, J. Biol. Chem. **56**:443 (June) 1923; Effect of Administration of Calcium Salts on the Inorganic Composition of Blood, J. Biol. Chem. **60**:327 (June) 1924.

10. Luckhardt, A. B., and Goldberg, B.: Preservation of the Life of Completely Parathyroidectomized Dogs, J. A. M. A. **80**:79 (Jan. 13) 1923.

11. Howland, J., and Marriott, W. McK.: Calcium Content of Blood in Infantile Tetany and Effect of Treatment by Calcium, Quart. J. Med. **11**:289 (July) 1918.

These results were again confirmed in this case. Early after the patient's admission we obtained more favorable results, however, than could be obtained later. At first, calcium chloride not only relieved the symptoms, but also increased the value of the serum calcium. We were never able later to reproduce this result. Chart 1 shows that enormous doses of calcium relieved the active symptoms, but did not alter the symptoms of latent tetany: the Chvostek sign, the electrical reactions, or the low serum calcium.

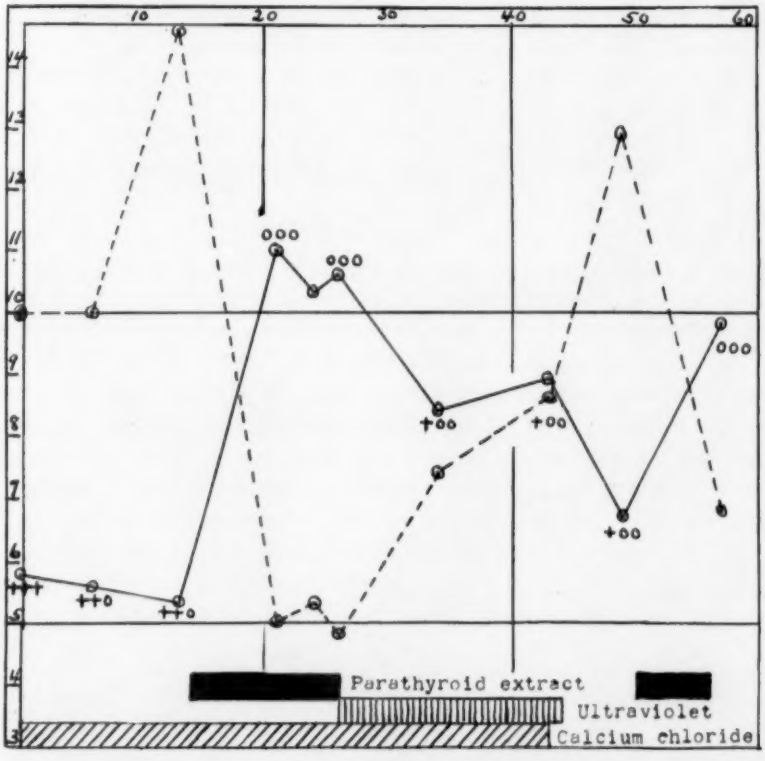

Chart 1.—The abscissae are day units. The ordinates are milligrams of calcium or phosphorus per hundred cubic centimeters of blood serum. The solid line is the calcium curve; the broken line is the phosphorus curve. This chart represents the effects of calcium chloride (60 grains [3.9 Gm.] daily), parathyroid extract and ultraviolet light (steadily increasing exposures to twenty-five minutes anteriorly and twenty-five minutes posteriorly) on the curves of calcium and phosphorus of the blood serum and the clinical signs of tetany. Three crosses indicate the presence of the Chvostek and Trousseau signs and the carpopedal spasm; two crosses and a circle, the presence of the Chvostek and Trousseau signs, and the absence of the carpopedal spasm; one cross and two circles, the presence of the Chvostek sign only; and three circles the absence of all signs.

Many observers [12] stated that ultraviolet light and cod liver oil have good effects in the treatment of infantile tetany and tend to raise the level of the serum calcium. In this child, we saw no effects at all when ultraviolet light was used, alone, or in conjunction with calcium or with cod liver oil (charts 1 and 2). This is in accord with the observations of Swingle and Reinhold [13] and Pincus, Peterson and Kramer [14] for ultraviolet light, and Jones [15] for cod liver oil, when treatment with these agents was started after parathyroidectomy had been done.

Collip [16] first reported the production and effects of a potent parathyroid extract, and later [17] summarized his own work and the results of others. He adhered to the view of MacCallum and Voegtlin that the reduction of the value of the serum calcium is the significant factor in tetany, and in support of this theory he pointed to the results of the new extract, not only in alleviating the symptoms of tetany, but also in raising the level of the serum calcium and maintaining it when experimentally used in animals.

Our results with Collip's extract support his claims to some extent. Charts 1 and 3 show examples of typical reactions obtained repeatedly following the administration of parathyroid extract (Collip). When maximal doses were given, the level of the serum calcium was raised to, or above, the normal, and all symptoms of tetany disappeared. We found that it was impossible to maintain the normal level for a long period of time on the extract alone, even if the dose was gradually increased. However, we believe that this was due to the progressive course of this case necessitating rapidly increasing doses of parathyroid hormone. Even when the action of the extract was enhanced by the use of calcium salts, cod liver oil and ultraviolet light, the level of the serum calcium tended to descend over a period of from three to four months, although the child was free from the convulsions of tetany and appar-

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12. Casparis, H., and Kramer, B.: Treatment of Active Infantile Tetany with Irradiations from Mercury Vapor Quartz Lamp, Bull. Johns Hopkins Hosp. **34**:219 (July) 1923. Hoag, L. A.: Treatment of Infantile Tetany with Ultraviolet Radiation, Its Effect on Clinical Symptoms and on Calcium Concentration in Blood Serum, Am. J. Dis. Child. **26**:186 (Aug.) 1923. Block, C. E., and Faber, F.: Light and the Antirachitic Factor, Am. J. Dis. Child. **30**:504 (Oct.) 1925.

13. Swingle, W. W., and Reinhold, J. G.: Effect of Ultraviolet Radiation upon Experimental Tetany, Am. J. Physiol. **75**:59 (Dec.) 1925.

14. Pincus, J. B.; Peterson, H. A., and Kramer, B.: J. Biol. Chem. **68**:601 (June) 1926.

15. Jones, J. H.: The Effect of the Administration of Cod Liver Oil upon Thyroparathyroidectomized Dogs, J. Biol. Chem. **70**:647 (Nov.) 1926.

16. Collip, J. B.: Extraction of a Parathyroid Hormone Which Will Prevent or Control Parathyroid Tetany and Which Regulates Level of Blood Calcium, J. Biol. Chem. **63**:395 (March) 1925.

17. Collip, J. B.: The Parathyroid Glands, Medicine **5**:1 (Feb.) 1926.

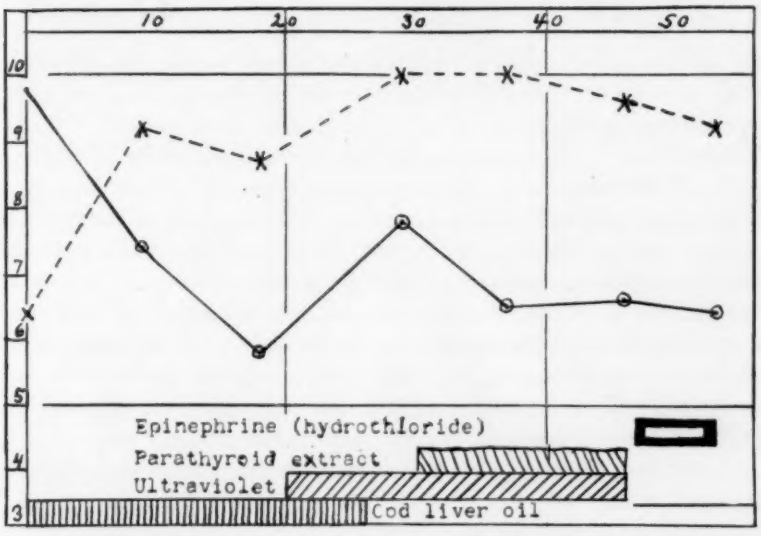

Chart 2.—This chart shows the failure of epinephrine hydrochloride (intramuscular injections of 1 cc. three times daily), cod liver oil (12 cc. daily), ultraviolet light (given with the mercury vapor quartz lamp; exposures steadily increased to twenty minutes anteriorly and twenty minutes posteriorly) and desiccated parathyroid extract (15 grains [0.9 Gm.] daily) to affect the values of the calcium and phosphorus of the blood serum.

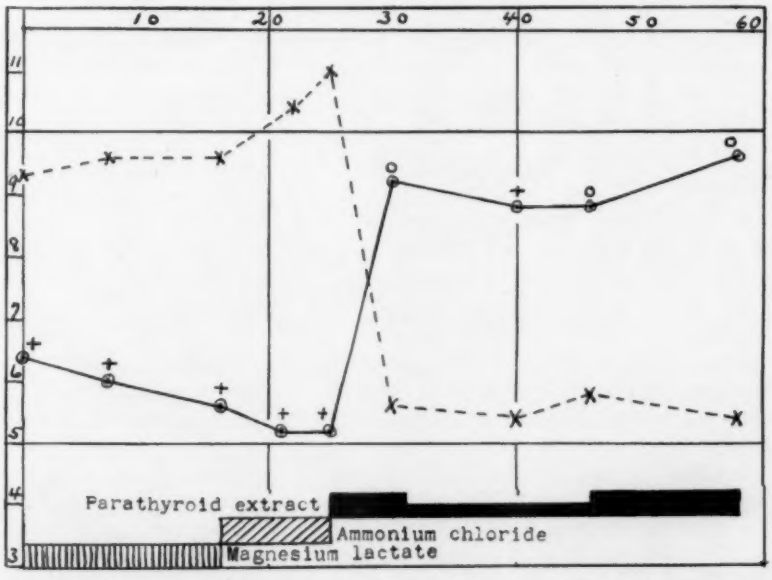

Chart 3.—This chart demonstrates the failure of the acid salts, magnesium lactate (60 grains [3.9 Gm.] daily) and ammonium chloride (120 grains [7.8 Gm.] daily), to change the values of the calcium and phosphorus of the serum. It also shows the varying results of minimal amounts (20 units and 10 units) of parathyroid extract over a period of thirty-three days.

ently in good condition. In July, 1928, it was found necessary to use from three to four times the amount of extract that was formerly needed to obtain results similar to those depicted.

An interesting feature was the action of the hormone on the value of the serum phosphorus. Kramer, Tisdall and Howland [18] found that the inorganic phosphorus in cases of infantile tetany showed great variation, but did not believe that the low phosphorus values were noteworthy, except when there was coincidental active rickets. Greenwald,[19] Elias and his associates,[20] Salvesen [9] and later Collip [17] all emphasized the increase in phosphorus in parathyroidectomized dogs, and Collip showed that the effect of the extract is to restore such an increased value to normal. Overdosage with the extract causes the phosphorus to rise again to very high levels in the preterminal period.

In our case, the inorganic phosphorus reached very high levels when the serum calcium was low, the phosphorus at one time mounting to the extraordinarily high value of 23.5 mg. Following the use of the extract, the phosphorus dropped to a normal level. Our curves of calcium and phosphorus values tend to prove the conception of Freudenberg and György,[21] which was later endorsed by MacCallum: [1]

$$\frac{(Ca^{\bullet\bullet})\ (HCo_3^-)\ (HPo_4^-)}{(H^{\bullet})} = K$$

Wenner [22] found that the administration of magnesium lactate, together with a diet of milk, caused abatement of the symptoms of tetany in parathyroidectomized dogs, and that when this treatment was continued for about forty days, the dogs were permanently cured and could be placed on a meat diet.

We found that this routine had no effect on our patient over a period of sixteen days.

Wilson [23] and his associates considered that there is a distinctive change in the acid-base equilibrium in tetany due to parathyroidectomy, and that the condition of partial alkalosis responsible for tetany is relieved by the production of acidosis. Many have advised the use of acids or

18. Kramer, B.; Tisdall, F. F., and Howland, J.: Infantile Tetany, Am. J. Dis. Child. **22**:431 (Nov.) 1921.

19. Greenwald, I.: Increase of Phosphorus in Tetany, J. Biol. Chem. **14**:369, 1913.

20. Elias, H., and Spiegel, E. A.: Tetany, Wien. Arch. f. inn. Med. **2**:447 (June) 1921. Elias, H., and Weiss, S.: Phosphorus Content of Serum in Titany, Wien. Arch. f. inn. Med. **4**:59 (April) 1922.

21. Freudenberg, E., and György, P.: Pathogenesis of Infantile Tetany, Klin. Wchnschr. **1**:222 (Jan. 28) 1922.

22. Wenner, W. F.: Prevention of Tetany by Oral Administration of Magnesium Lactate, Am. J. Physiol. **81**:392 (July) 1927.

23. Wilson, D. W.; Stevens, T., and Janney, J. H.: The Effect of Acid Administration on Parathyroid Tetany, J. Biol. Chem. **21**:169, 1915.

acid-producing salts in the various types of tetany. Following the work of Haldane [24] with ammonium chloride, McCann,[25] Wenner,[26] and others have advocated its use in tetany.

We gave our patient 120 grains (8 Gm.) of ammonium chloride per day for nine days and produced a compensated acidosis, but effected no change at all in the symptoms of tetany or in the value of the serum calcium (chart 3).

An ordinary commercial preparation of dried parathyroid extract was also shown to be of no value (chart 2).

Some interesting symptoms exhibited by this child deserve special mention. At no time did we observe any changes in bone other than those of healed rickets, acquired during infancy. The teeth were soft and broke easily, and seemed to be deficient in enamel. The finger and toe nails were curiously malformed, being ridged and soft and almost of the consistency of waxed paper. At one time, when she was unobserved for a period of two months, she developed large corneal ulcerations, which healed slowly under adequate treatment for the tetany. The condition of the teeth in tetany was noted by Erdheim, while the changes in the nails and the eyes were described by Sainton-Poron.[27]

On Nov. 22, 1928, the patient developed fever, severe diarrhea and an infection of the upper respiratory tract. The leukocyte count was 13,200, and the hemoglobin was 72 per cent. For the next three days, the temperature curve was of the septic type, and a diagnosis of bronchopneumonia was made on November 24. During the next twenty-four hours, she rapidly became worse, and she died on November 25. She was treated by transfusions of blood, and various drugs were used for stimulation.

Previous to the illness, the serum calcium was 7.5 mg. per hundred cubic centimeters., and the serum phosphorus was 11.3 mg. per hundred cubic centimeters. The day before death, the calcium and the phosphorus determinations were 7.9 and 9.8 mg., respectively. Just prior to death, a sterile solution of calcium lactate was injected into the ventricular chamber of the heart. Following death, blood was removed from the heart and tested for calcium, but the result was 19.5 mg. per hundred cubic centimeters, showing that stagnation had occurred. However, a postmortem lumbar puncture was done, and the calcium and the phosphorus in the spinal fluid were 6.7 and 8 mg. per hundred

24. Haldane, J. B. S.: Experiments on the Regulation of the Blood's Alkalinity, J. Physiol. **55**:265 (Aug.) 1921.

25. McCann, W. S.: A Note on the Use of Ammonium Chloride in Tetany, Proc. Soc. Exper. Biol. & Med. **19**:393, 1921.

26. Wenner, W. F.: Prevention of Tetany by Oral Administration of Ammonium Chloride, Am. J. Physiol. **81**:612 (Aug.) 1927.

27. Sainton-Poron: Rev. neurol. **29**:442, 1922.

cubic centimeters, respectively. These values represent better the actual concentrations of calcium and phosphorus in the body fluids post mortem.

Unfortunately, further studies could not be made, since permission for autopsy could not be obtained.

Previous to the patient's death, studies were under way to determine the exact effect of parathyroid hormone on the metabolism of calcium, phosphorus and other ions. Such studies had previously been reported by Greenwald and Gross [28] for parathyroidectomized dogs, and by Hoag et al.[29] and Shohl et al.[30] for infantile tetany. Other studies were in progress to determine the effect of irradiated ergosterol.

SUMMARY

1. A case of chronic tetany in a child of 7 years is reported. The onset occurred when she was 4 years of age and coincided with the appearance of a chronic oral infection. The organism causing the latter was not identified, but was of the *Monilia* group.

2. Despite the use of all the well known agents advised for tetany, the child became progressively worse.

3. The results of several types of therapy are shown with special reference to their effects on the values of the serum calcium and phosphorus and on the symptoms of tetany. The only one of this group shown to have any pronounced effect is parathyroid extract (Collip).

4. All agents advocated as clearing up ordinary oral infections proved of no use in this case.

5. It is suggested that this case more nearly resembles the tetany produced by parathyroidectomy than ordinary infantile tetany.

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28. Greenwald, I., and Gross, J.: Effect of Administration of Parathyroid Extract on Excretion of N, P, Ca, and Mg with Remarks on Solubility of Calcium Phosphate in Serum and on Pathogenesis of Tetany, J. Biol. Chem. **66**:217 (Nov.) 1925.

29. Hoag, L. A.; Rivkin, H.; Weigele, C. F., and Berliner, F.: Effect of Potent Parathyroid Extract on Calcium Balance in Infants, Am. J. Dis. Child. **33**:910 (June) 1927.

30. Shohl, A. T.; Wakeman, A. M., and Shorr, E. V.: Effect of Parathyroid Extract on the Mineral Metabolism in Infantile Tetany, Am. J. Dis. Child. **35**:392 (March) 1927.

THE FLUORESCENT PROPERTIES OF RINGWORM OF THE SCALP AS AN AID IN DIAGNOSIS*

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Ultraviolet light when passed through a glass filter containing nickel oxide will cause certain substances to fluoresce in a characteristic manner. Wood's glass, invented by Prof. R. W. Wood of Johns Hopkins University, or Chance's ultraviolet glass, developed by Chance Brothers of Smethwick,[1] is a deep violet nickel oxide glass, which transmits only the shortest rays of the visible spectrum and between 70 and 80 per cent of the ultraviolet rays in the region between 3,000 and 4,000 angstrom units.[1] Solutions of eosin, quinine, quinidine, zinc sulphate and sodium salicylate give a distinctive fluorescence under such a light. Petrolatum, hydrous wool fat, mustard dried on cloth,[1] dried blood and semen,[2] and cerumen from the ear can be detected under Wood's light by their fluorescent properties. The normal skin and hair fluoresce faintly, the finger-nails fluoresce rather more brightly with a bluish tinge, and the teeth appear a brilliant white in contrast with dark areas caused by fillings, false teeth or large caries. In marked contrast with normal hair, hairs infected with ringworm fluoresce brilliantly with a glowing green. Areas on the scalp containing broken off hairs infected with ringworm in the mouths of the follicles also fluoresce a brilliant green.

The appearance of tinea tonsurans under Wood's light was first described by Margarot and Deveze in 1926.[3] A year later Vigne[4] presented a method of examination, together with the satisfactory results of the use of the light in eradicating ringworm from an orphange in which it had been present for thirty years. In 1927, Roxburgh demonstrated the use of the light before the Section of Dermatology of the

* Submitted for publication, Feb. 12, 1929.

* From the Department of Pediatrics, University of California Medical School.

1. Hill, N. G.: Wood's Glass and the Detection of Ringworm by Its Fluorescent Properties, Brit. J. Child. Dis. **25**:54, 1928.

2. Cleveland, D. E. H.: "Wood Light" in Dermatologic Diagnosis with Special Reference to Ringworm, Arch. Dermat. & Syph. **18**:368 (Sept.) 1928.

3. Margarot, J., and Deveze: Aspect de quelques dermatoses en lumière ultraparaviolette. Note preliminaire, Bull. Soc. d. sc. méd. et biol. de Montpellier **7**:375, 1925.

4. Vigne, P.: Empoli de la lumière ultra-violette de Wood pour le dépistage des teignes, Marseille méd. **63**:32 and 1765 (Nov.) 1926; Utilization de la lumière de Wood pour l'examen et le dépistage des teignes tondantes et du favus, Presse méd. **35**:339 (March) 1927.

Royal Society of Medicine,[5] and wrote a clinical note[6] on its employment in hospital and private practice. He stressed the use of the light in ascertaining the results of epilation, in preventing recurrences and in deciding whether or not the contacts of the patient infected with ringworm were infected. The practical use of the light in the service of a large hospital was discussed by Hill,[1] who favors this method of routine examination for admission to the wards because of its accuracy and saving of time. Twenty or thirty patients may be examined with the light[1] in an hour. Recently Cleveland[2] gave an account of his experiences in the use of the light as a differential diagnostic agent and said that it was a reliable means of detecting infections with ringworm. To date, American medical literature contains only one reference to this method of diagnosis.

TECHNIC AND EQUIPMENT

The source of the ultraviolet light may be a mercury vapor air cooled light, a water cooled lamp (Kromayer), the tungsten arc or carbon arc light.[6] The ordinary carbon arc light, such as is used for the projection of lantern slides, may also be used.[6] Wood's glass is attached to any of these lights by the adaptors or shutters which come as standard equipment with most of the commercial lights, or, if the aperture transmitting the light is small, the glass may be attached by means of adhesive tape.

The examination is carried out in a darkened room, the source of the Wood light being held 3 or 4 inches (7.6 or 10.16 cm.) from the head to be examined. The hairs infected with tinea will fluoresce a brilliant green. Hairs infected with the microspora fluoresce the most brilliantly,[7] those with trichophyton somewhat less and those with favus[8] only faintly. In favus, the crusts must be removed, and at times a magnifying glass must be used to see the fluorescent hairs about the edge of the lesion.

Ringworm of the body in most cases does not fluoresce under the Wood light. Sibley,[9] however, described the fluorescence of a tinea circinata of the forehead, and Cleveland[2] reported typical fluorescence of fungus infiltrated skin on the scalp and forehead. Epidermophyton inguinale, sycosis, psoriasis,[10] erythrasma and pityriasis versicolor[8] do not fluoresce under the Wood light.

If ointments containing petrolatum or hydrous wool fat have been used on the scalp previous to the examination, these should be removed with ether or soap and

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5. Roxburgh, A. C.: Demonstration of the Detection of Ringworm Hairs on the Scalp by Their Fluorescence Under Ultraviolet Light, Proc. Roy. Soc. Med. **20**:1200 (March) 1927.

6. Roxburgh, A. C.: Clinical Note—the Detection of Ringworm Hairs on the Scalp by Their Fluorescence Under Ultraviolet Light, Brit. J. Dermat. **39**:351, 1927.

7. Vigne (footnote 4, second reference). Sibley, in discussion of article by Roxburgh (footnote 6).

8. Margarot and Deveze (footnote 3). Vigne (footnote 4, second reference).

9. Sibley, in discussion of article by Roxburgh (footnote 6).

10. Cleveland (footnote 2). Margarot and Deveze (footnote 3).

water, as they have fluorescent properties that interfere with the examination for ringworm. Tincture of iodine, solutions of mercurochrome, gentian violet and mild silver-protein when present on the infected hairs, inhibit the normal fluorescence.

ILLUSTRATIVE CASE REPORTS

The use of the lamp in a clinic practice may be illustrated by the following cases:

J. B., a boy, aged 6 years, with ringworm of the scalp, was referred to me after epilation of the infected areas by local treatment. Three unsuccessful attempts to recover infected hairs from the margins and centers of such areas had resulted in the opinion that the patient was cured. Under the Wood light, the epilated areas and their margins were nonfluorescent. Scattered through the scalp, however, were individual brilliantly fluorescent hairs. These hairs, when extracted and prepared, showed under the microscope a typical trichophyton. All fluorescent hairs were extracted, and the patient was seen again at weekly intervals for three weeks, with no evidence of recurrence.

D. P. and F. P., sister and brother, came to me for consultation because they had been excluded from school with a suggested diagnosis of favus. Crusts were present on the heads of both, and the boy, in addition, showed a few areas containing broken off hairs. After the crusts were removed, both children were examined under the light. The girl did not show any fluorescent hairs. The boy showed several brilliantly fluorescent areas in which the hairs were broken off. Areas in which only crusts had been present showed no fluorescence. A diagnosis of impetigo contagiosa was made for both children, with the addition of ringworm of the scalp for the boy. The brilliance of the fluorescence of the hairs infected with ringworm on the boy's head immediately excluded favus and suggested a microspora or a trichophyton. Microscopic examination showed a trichophyton. The time necessary for the examination of both children under the light and for the arrival at an accurate diagnosis and prognosis was ten minutes.

COMMENT

The use of this diagnostic aid in ringworm of the scalp recommends itself because it is specific [1] and because it is easy to apply. Mercury vapor and carbon arc lights are now available in most offices and hospitals, and Wood's glass is readily obtainable and easily attached to any of the lights. This method of examination has been used in the outpatient service of the department of pediatrics of the University of California for the study of all suspected cases of tinea of the scalp, and has been found much more expedient than the older method of microscopic examination of the infected hairs. With the light, one is able to detect isolated infected hairs and to remove them, thus preventing recurrences and spread. After epilation or treatment by any method, one can readily make a careful check of the efficiency of the treatment, and if there are any infected areas still present or any new areas developing, these can be seen and treated before recurrence. The Wood light is also an accurate means of rapid differential diagnosis in doubtful cases and of great value in determining when a patient is cured.

THE ANTIDIURETIC EFFECT OF SOLUTION OF PITUITARY DROPPED INTRANASALLY IN A CASE OF DIABETES INSIPIDUS*

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A. E., a girl, aged 9 years, first appeared in the clinic in April, 1927, with the complaint of increased thirst, frequency of urination and marked malnutrition. The past and family histories were essentially unimportant. The present illness began in October, 1925, at which time she had to be removed from school because of polydipsia and polyuria.

Examination.—Physical examination showed an extremely malnourished and markedly underweight child (fig. 1). The weight was 38 pounds (17.2 Kg.). She seemed rather dull and apathetic, and did not take much interest in her surroundings. The skin was dry and loose and the bladder was distended to the umbilicus; otherwise, the results of the physical examination were negative. The results of the special examinations, as of the fundus and visual fields, and the results of laboratory examinations, including the chemical analysis of the blood, the Wassermann test of the blood and tests of the kidney function, were all negative. An x-ray picture of the sella showed it to be normal. The urine showed: twenty-four hour output, 4,800 cc.; albumin, negative, and sugar, negative; the microscopic examination was negative.

Course.—The patient was sent to the hospital, where the diagnosis of diabetes insipidus was confirmed, and studies on the administration of solution of pituitary were begun. The general course and effect on the urinary output and intake are shown in figure 2. The daily excretion, shown in the upper section of figure 2, showed an output of 4,800 cc. in twenty-four hours, with a corresponding intake of 3,200 cc. of fluid, as shown in the second section. The lower section of figure 2 represents the weight. Solution of pituitary was administered intramuscularly, 6 minims (0.36 cc.), three times a day, which soon reduced the urinary output to around an average of 1,600 cc. daily, while the intake remained around 1,500 cc. The patient was discharged in June, and returned in July, 1927, saying that solution of pituitary had not been given because of the difficulty of hypodermic administration in the home. She was again sent to the hospital, where solution of pituitary from the posterior lobe, 6 minims (0.36 cc.), was given three times a day intramuscularly. This was followed by a reduction of the output and intake, as shown in figure 2. In September, the urinary symptoms remained within normal limits and the child began to show great increase in weight, in five months gaining 23 pounds (10.4 Kg.). In September, obstetric solution of pituitary, 15 minims (0.92 cc.), was given by a medicine dropper with the patient's head fully extended so that the solution would come in contact with the mucous membrane of the nasopharynx. Doses of 15 minims (0.92 cc.) four times a day, were given in this manner and are shown by the broken line at the top of figure 2. This proved to have the same effect as the solution of pituitary given hypodermically.

The patient continued to show a normal intake and output until January, 1928, when she developed diphtheria; she was sent to the hospital for contagious dis-

* Submitted for publication, March 5, 1929.

cases, where pituitary extract was not administered. Figure 2 shows the rapid rise of the urinary output, from 1,600 to 3,500 cc. during her stay in the hospital. This rise was accompanied by a corresponding fluid intake, as shown in figure 2. When the patient was transferred from the contagious ward, solution of pituitary was again administered by the nasal route, causing the output and intake to fall to normal limits. She was then sent home in May, 1928, where solution of pituitary was administered three times a day by the medicine dropper. She returned for observation in September, 1928, showing a great increase of the fat tissue of the body; she had become obese (fig. 3). At that time the child weighed 82 pounds (37.2 Kg.), a gain of 42 pounds (19 Kg.) in one and a half years. Because of

Fig. 1.—A. E., April, 1927; weight, 38 pounds.

this change, the doses of solution of pituitary were discontinued during the month of October, which caused a return of the symptoms of diabetes insipidus. The administration of solution of pituitary was again resumed in November, 1928, causing a disappearance of the polyuria and polydipsia.

COMMENT

This case is presented: (1) as a case of diabetes insipidus of unknown etiology with response to solution of pituitary; (2) as a case of diabetes insipidus responding to treatment with solution of pituitary

administered by the intranasal route, and (3) because of the unusual gain in weight.

It has been demonstrated that many cases of diabetes insipidus have shown response to solution of pituitary. The necessity for repeated hypodermic injections of solution of pituitary from the posterior lobe into patients with diabetes insipidus has stimulated investigators to devise some method of administration less painful and more convenient than that of the hypodermic administration. In 1922, Blumgart [1]

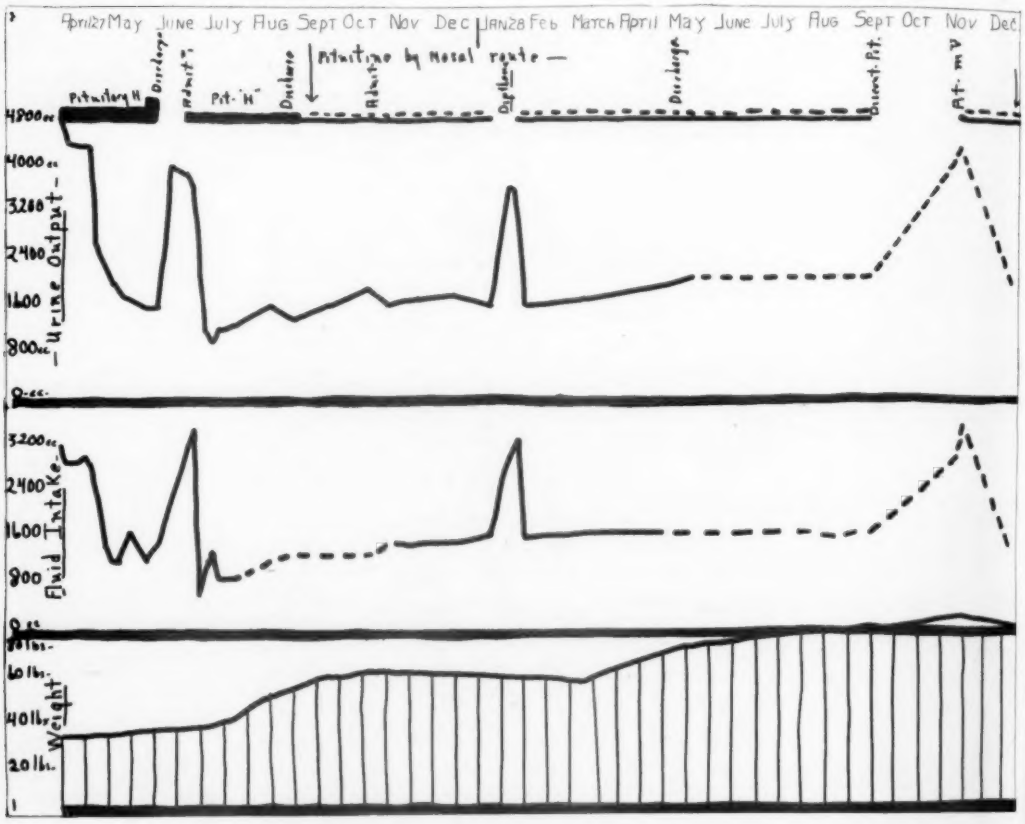

Fig. 2.—The upper curve represents fluctuations in urinary output as the result of administration of epinephrine; the center curve, fluid intake; the lower curve, weight. At the top of the chart the solid line represents the administration of solution of pituitary hypodermically; the solid and broken line, intranasally. In the curves, the broken line represents the output and intake as measured when the patient was out of the hospital; the solid line, as measured when the patient was in the hospital.

1. Blumgart, H. L.: Antidiuretic Effect of Pituitary Extract Applied Intranasally in a Case of Diabetes Insipidus, Arch. Int. Med. **29**:508 (April) 1922.

demonstrated that solution of pituitary applied intranasally checked both the polyuria and the polydipsia as effectively as hypodermic injections. In 1928, Blumgart [2] again reported eight cases with response to intranasal administration of solution of pituitary from the posterior lobe. In these cases solution of pituitary was applied by atomizer or by the application of a cotton swab soaked in from 0.5 to 1 cc. of the solution and placed against the nasopharyngeal membrane, where it was allowed to remain until the next application was to be made. In the case here reported,

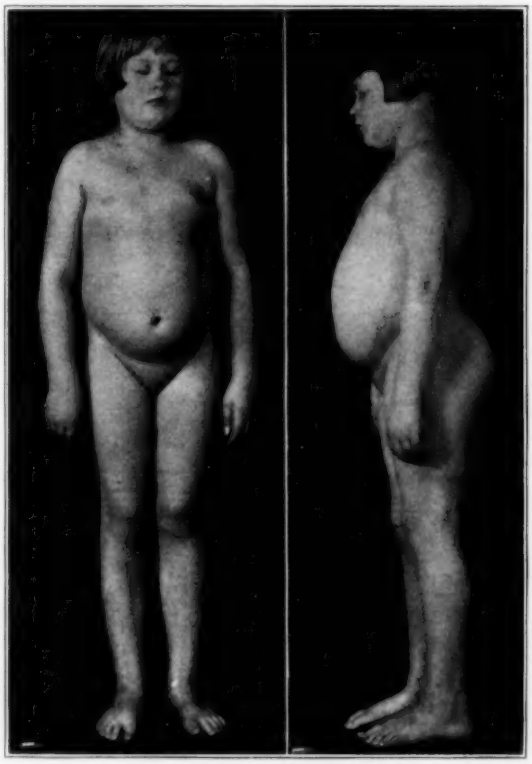

Fig. 3.—A. E., December, 1928; weight, 82 pounds.

equal results were obtained by dropping the solution of pituitary by means of a nose dropper into the nose of the patient after the head was extended over the edge of the table or bed so that the drops came into contact with the nasopharyngeal mucous membrane. Attempts to cause absorption of the solution of pituitary by other routes than by the mucous membrane of the nose, as by the stomach on administration by

2. Campbell, J. R., and Blumgart, H. L.: The Treatment of Diabetes Insipidus with Posterior Lobe Extract Applied Intranasally, Am. J. M. Sc. **176**:769 (Dec.) 1928.

mouth or by rectum, have been unsuccessful. Blumgart [3] has shown that peculiar physiologic absorptive properties are related to the unusual anatomic structure of the nasopharynx in that it possesses free nerve endings that penetrate through the mucous membranes. Solution of pituitary is absorbed here by way of the lymphatics of the olfactory nerve cells and from thence is transferred to the systemic lymphatics.

The unusual gain in weight, as shown in figures 1 and 3, is of further interest in the consideration of this case, when one considers the various functions of the pituitary gland. The following is an outline of the pituitary functions:

A. Anterior Lobe
- Glandular structure
- Controls—tissue growth (skeleton and sex)
  - (*a*) Hypersecretion—gigantism
  - (*b*) Hyposecretion—infantilism

B. Intermediate lobe
- Nervous structure

C. Posterior lobe
- Nervous structure
- Controls—metabolic processes
  - Secretion of urine
  - Sugar tolerance
  - (*a*) Hyposecretion—drowsiness
    - Slow pulse and respiration
    - High sugar tolerance
    - Increase of fat deposits
  - (*b*) Hypersecretion—opposite of hyposecretion, as loss of weight, etc.

When the patient was first seen, showing extreme emaciation and symptoms of diabetes insipidus, one was forced to consider a hypersecretion of the posterior lobe. Yet, the administration of solution of pituitary caused the symptoms of diabetes insipidus to disappear and the patient to appear as having a case of hyposecretion as indicated by the marked increase in fat deposits. When one considers that the functions of the pituitary gland and its relation to diabetes insipidus are not definitely known, one can assume that the disease may be a combination of all structures of the pituitary gland. At any rate, I assume that in this particular case I have been dealing with a hypersecretion of the gland which has finally lapsed into insufficiency and a corresponding Fröhlich's syndrome.

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3. Gargle, S. L.; Gilligan, D. R., and Blumgart, H. L.: The Antidiuretic Effect of Oxytocic and Pressor Principles of Posterior Lobe of the Pituitary, New England J. Med. **198**: 169, 1928. Blumgart, H. L.: A Study of the Mechanism of Absorption of Substances from the Nasopharynx, Arch. Int. Med. **33**:415 (April) 1924.

SUMMARY AND CONCLUSIONS

1. A case of diabetes insipidus with response to the nasal administration of solution of pituitary has been studied.

2. The intranasal application of solution of pituitary from the posterior lobe given by nasal drops seems as efficacious as other methods heretofore described.

3. Attention is drawn to the unusual gain in weight, and comments are made concerning the relation of this result to the functions of the pituitary gland.

BONE DYSTROPHY

WITH CHARACTERISTICS OF LEONTIASIS OSSEA, OSTEITIS DEFORMANS AND OSTEITIS FIBROSA CYSTICA IN A CHILD. A SUGGESTION AS TO THE INFLUENCE OF THE CENTRAL NERVOUS SYSTEM *

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NEW YORK

History.—J. S., a boy of Russian Jewish parentage, aged 11 years, was brought to the outpatient department of the New York Neurological Institute, March 15, 1926, with the complaint of blindness. He said that he did not remember ever having seen with his right eye. At the age of 6, when he was first sent to a New York public school, his defective vision was called to the attention of his parents; at this time, vision in the left eye was also apparently failing. He continued in the public school for normal children for another two years in spite of his defective vision, but at 8 years of age was unable to continue because of complete failure of vision and was transferred to the school for the blind. (He was attending this school when he was last seen.) In addition to the blindness, a secondary complaint was enlargement and deformity of the face and skull. When he was 1 year old, his mother first noticed that the right side of the face was larger than the left. This became more and more pronounced, and the forehead, right side of the head and occiput became involved in a massive deformity. It is thought that there has been a slow and steady growth and accentuation of this formation even in the six months preceding examination at the institute. In addition, there was a minor complaint of slight deafness. No abnormality of any other part had been noticed by the patient or by his mother, although other changes in contour and bodily proportion were found. There had never been any pain or tenderness about the head, face, limbs or body. There had never been any muscular weakness or incoordination or any other condition that had interfered with normal activities. He was handicapped only by blindness.

The boy was the last of four children. His mother's pregnancy was normal; labor lasted two hours and was relatively easy; delivery was normal. His early development was normal. At 3 years of age he had a convulsion while he was apparently in his usual good health. This was preceded by malaise for about half an hour, during which he was lying down, when he suddenly became unconscious and cyanotic. There was generalized rigidity for a few seconds, then generalized clonic convulsions, with stertorous breathing, salivation and urination. The attack lasted about a minute and a half, and after a short period of stupor, the boy resumed his normal activities and seemed well. A physician arrived after the attack was over and attributed it to a gastro-intestinal disturbance. The boy never had another similar attack nor any other episode that might be interpreted as a petit mal attack. Aside from measles at 6 years of age, he has had excellent general health, and there has been no bodily disturbance except those associated with the condition already described. Careful inquiry revealed nothing unusual—no abnormal intake and output of water, disturbance of sleep, dietary variations, etc.

* Submitted for publication, Dec. 6, 1928.

When the boy was 6 years old, his mother took him to the outpatient department of Mount Sinai Hospital on account of failing vision and the deformity of the face and skull. According to her statement, roentgen examination was made. I have not been able to find any record of his visit to the hospital at that time, nor have the x-ray plates been found there. The mother had the impression that no diagnosis was made and said that she was told that he was too young for any action to be taken. She was told that he should remain under observation, but she never took him back. After his preliminary examination in the outpatient department at the Neurological Institute, he was admitted to the ward there in

Fig. 1.—Enlargement and deformity of right side of face and skull, arm and leg, suggesting hemihypertrophy but due to disease of the bone.

Dr. Kennedy's service, where some observations were made. Later, he was admitted to the ward at Bellevue Hospital, Dr. Kennedy's service, for a few days; then his mother took him home. Every effort was made to get him back for reexamination, but his mother refused any further contact, probably because no cure resulted from his two brief hospital courses.

The boy's parents were Russian Jewish immigrants. Nothing is known in detail of the grandparents or collaterals. The boy's mother said that as far as she knew they were all normal, hardworking people. The father was active and robust; he was not syphilitic and had never been ill as far as could be ascertained. He was a housepainter and worked at this trade for many years up to his death,

which resulted from injuries sustained when he fell from a scaffold one and a half years before the boy was first seen. The mother was in good health at the time the boy was examined. She said that she had always been well. She had had four normal pregnancies and four children normal at birth. The first born was a girl, aged 20, well developed and healthy, a school teacher. The second was a girl, aged 18, also a school teacher. The third was a boy, aged 17, in his first year at City College. The patient was the fourth and last child.

Physical Examination.—The boy was bright and alert; his behavior was normal, and was modified only by his blindness and partial deafness. His intelligence

Fig. 2.—Asymmetry and deformity of face and skull showing distortion of features and proptosis.

quotient was 142 according to the Terman modification of the Binet-Simon tests, as reported by Miss Tallman, psychologist at the Neurological Institute. He impressed one as being taller and heavier than the average 11 year old boy, although there were no actual measurements in the records. He was rather pale; a few acneiform papules and pustules were scattered over the forehead. Grossly, the right side of the body seemed to be larger than the left; when this boy first appeared in the clinic, it was thought that he had hemihypertrophy, but there was actually no true enlargement of all tissue elements on one side of the body. The right arm was apparently longer than the left, and the right leg was obviously longer than the left. There was an abnormal angulation of the femur and pelvis

and at the knee and ankle. There was a large bony mass that seemed to be an exaggeration of the normal contour over the mesial aspect of the lower end of the femur. Asymmetry and deformity of the face and skull were conspicuous, being most marked on the right side. This was found to be due to bone-hard masses which were not sharply circumscribed but which merged gradually into relatively normal skull contours. There was proptosis on both sides, more on the right; the right eyeball was dislocated downward and laterally. The buccal cavity presented an interesting situation. The right hard palate and upper alveolar process encroached on the normal space within the mouth. The teeth were widely spaced and irregularly placed. Normal approximation of the upper and lower

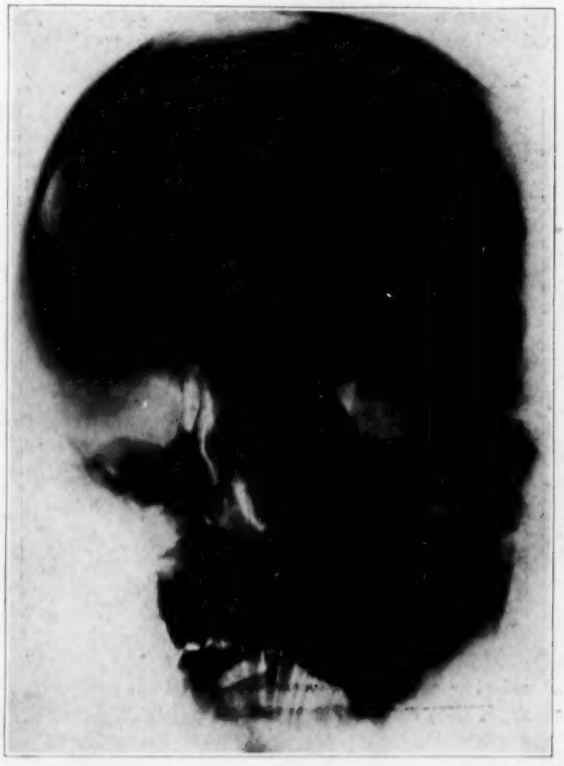

Fig. 3.—Skull of same patient showing dense overgrowth of bones of right side of face associated with leontiasis ossea.

dentures was not possible, and the boy could not close his mouth and keep it closed naturally and comfortably. On palpation of the region between the hard palate and the external part of the cheek on the right side, it was evident that tremendous thickening had taken place. The profile presented a saddle nose effect because of the disappearance of the normal depression between the side of the nose and the cheek. There was an especially large, bone-hard mass over the right occiput which protruded conspicuously and showed a sharp delimitation where it sprang from the nape of the neck and mastoid region. The view of the back of the head showed the deformity described earlier and, in addition, a so-called birth mark, or nevus, an interesting skin disturbance worth noting.

Neurologic Status.—There was no disturbance in smell. Bilateral primary optic atrophy was present. The patient had no perception of light in the right eye and only faint perception of light in the left eye; all movements of the extraocular muscles were well performed; there was no ptosis (the boy is looking downward in figure 2); he had no oculomotor weakness; the uncontrolled drifting movements of the blind were present, but no true nystagmus. There was partial nerve deafness on the right side. Stimulation of the vestibular apparatus showed lessened irritability on both sides. All other cranial nerves were functioning normally. The general motor, reflex and sensory conditions were normal. There were no cerebellar signs.

Laboratory Examination.—Nothing unusual was found. There was a mild secondary anemia. The blood chemistry was within normal limits; a dextrose

Fig. 4.—Lateral view of skull shown in figure 3, showing the fluffy, motheaten appearance associated with osteitis deformans (Paget's disease). There are areas of decreased density in the frontal bone indicating formation of cysts.

tolerance test showed normal function. The cerebrospinal fluid was normal. Basal metabolism was within normal limits.

X-Ray Report (Dr. Schwartz, Roentgenologist at New York Neurological Institute).—"Examination of the skeleton shows the bones of the skull to be involved in a profound pathological process which is much more marked on the right side. This has resulted in considerable bony overgrowth involving particularly the frontal bone and the bones of the base as far back as the foramen magnum. It involves the bones of the face and the occipital bone, to a less extent the occipitoparietal. The overgrowth is dense, excepting in the frontal bone, which shows areas of decreased density not unlike small cystic formations. Aside from the cystic areas noted, the bone has the appearance usually associated with leontiasis ossea. The sella is small; whether originally larger than it is

at present it is difficult to state with accuracy, due to the overgrowth of bone mentioned which involves the sella. It is now of small capacity as compared with the size of the skull. The petrous portion of the temporal bone is beginning to be involved so that hearing will probably be gradually lessened. The bone is comparatively free of changes as yet. The vault of the skull shows the typical fluffy, motheaten appearance of Paget's disease.

"Examination of the vertebrae shows them to be comparatively normal in outline as far as the lumbar region. The ribs are as yet free of any definite pathological change. The right humerus, radius, to a less extent, the ulna, also the phalanges and metacarpals, the right femur, tibia and fibula, the right half of the pelvis and the lumbar vertebrae are all involved in a pathological process

Fig. 5.—Right humerus and radius showing fibrous bony overgrowth with thinning of medullary canals and intact cortex. The same process is seen in the ulna to a less degree. The articular surfaces and the epiphyses are uninvolved.

which is characterized by a fibrous bony overgrowth with, in the shafts of the long bones, a thinning of the cortex and enlarging of the medullary canals, but the cortex it still intact. In numerous places in the medullary canal there are areas of decreased density suggesting cystic formation. The epiphyses are uninvolved. The articular surfaces, except in the right hip, are still uninvolved. The changes noted suggest strongly two pathological processes: Paget's disease and osteitis fibrosa cystica. Paget's is, of course, rarely found in the first age period, whereas osteitis fibrosa cystica is a definite disease of the first age period. It seems highly probable that this is a case of atypical juvenile Paget's disease, with

the probability of its also being osteitis fibrosa cystica. Pathological fractures will probably occur before long as the cortex of the right femur is already very thin."

Dr. Schwartz stated that the changes were more marked on the right side. He amended this to say that he saw no pathologic changes in the bones on the left side except in the skull and minor extension beyond the midline in the sacrum.

COMMENT

This boy had, in brief, an osseous disease with multiple lesions in the long bones of the right side of the body and sacrum mainly on the right side and in all the bones of the skull. It started, if the observation

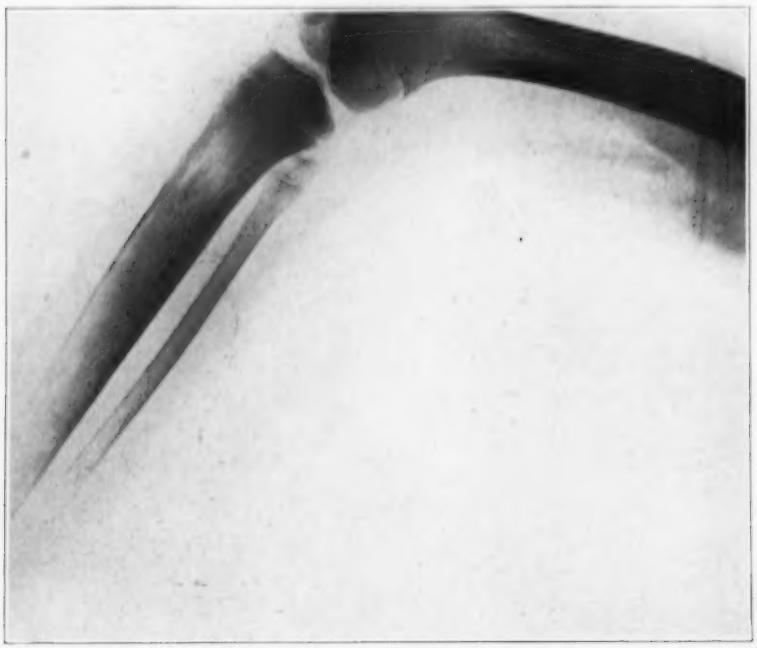

Fig. 6.—Right femur, tibia and fibula showing the same process with areas of decreased density suggesting cyst formation and threatening pathologic fracture.

of the boy's mother is accepted, in the right malar bone during the first year of his life and with slow, painless progression produced enormous thickening and deformity of the bones of the face and skull and gradual encroachment on the optic foramen and the internal auditory meatus, with resulting primary atrophy of the optic nerve and deafness. The involvement of the long bones and pelvis was in all likelihood a later development. The x-ray films show extremely dense impenetrable masses in the region of the earlier involvement, i.e., the bones of the skull. In the field of later development are areas of rarefaction, osteoporosis, thinning of the cortex and what is interpreted as repair

process. In areas in the skull there is the fluffy, motheaten appearance associated with Paget's disease. The case presents features definitely associated with leontiasis ossea, or diffuse hyperostosis of the bones of the skull and face; with Paget's disease, or osteitis deformans, and with osteitis fibrosa cystica of von Recklinghausen.

The literature on these topics and on allied subjects into which one finds oneself drawn is large. In addition to reports of typical, classic cases of all three—of leontiasis ossea, as first described by Virchow,

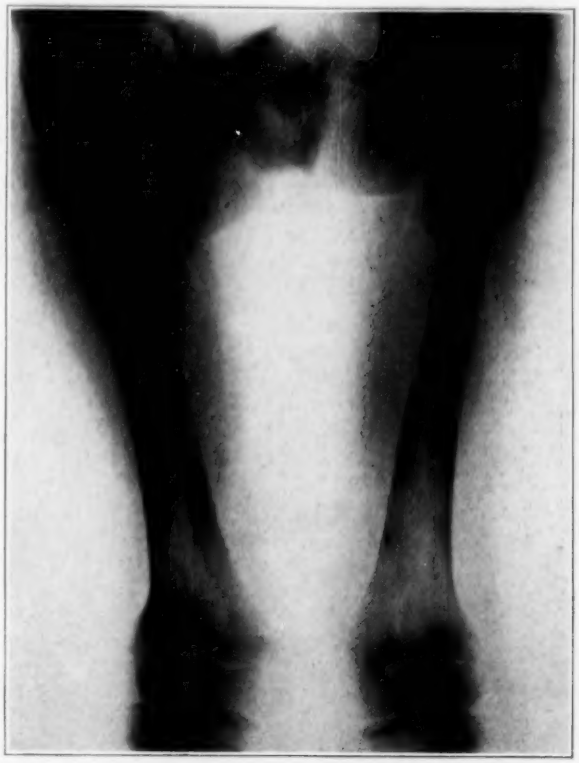

Fig. 7.—Appearance of both femurs.

osteitis deformans of Paget, and osteitis fibrosa cystica of von Recklinghausen—there are many cases in some respects atypical which show an incomplete picture, unusual symptoms and unusual localization, cases in which the patients are outside the classic age limits, and cases which suggested transitional stages in any one or two of the diseases. These have stimulated many reviews of the literature and analyses with attempts at classification, which will not be detailed here. In the literature of leontiasis ossea, or diffuse hyperostosis of the bones of the face and skull, the disease process is described as essentially an

excessive proliferation of bone in the skull and face with encroachment on the foramina and fossae, nipping off cranial nerves and giving corresponding symptoms and signs. It is described classically as occurring at any age, less frequently during the first decade. Bockenheimer,[1] in a review of twenty-five cases and a report of five of his own, mentioned involvement of the fibula in one.

The classic picture of the disease described by Paget to which he applied the term osteitis deformans need not be restated here. Of the

Fig. 8.—Pelvis of same patient. The vertebrae are normal to the lumbar region. The lumbar vertebrae, sacrum, right ilium, right hip joint and right femur are involved.

three conditions under discussion, this has been stretched to include the greatest variety of atypical pictures. Many cases of juvenile Paget's disease are on record. There are cases of Paget's disease with involvement of only one or two bones, in the tibia, in the jaw bone, in the

1. Bockenheimer: Ueber die diffusen Hyperostosen der Schadel und Gesichts Knocken S. Osteitis Deformans fibrosa (Virchow's Leontiasis ossea), Arch. f. klin. Chir., vol. 85.

clavicle, interpreted as early stages of Paget's disease; of Paget's disease with formation of cysts; of the diseases with fractures, which by the way, do not unite, in contrast with fractures in osteitis fibrosa cystica, which are frequent and which do unite. There are a few cases of so-called Paget's disease involving only the malar bone and one the hard palate, suggesting rather the diffuse hyperostosis of bone of the face and skull, or leontiasis ossea. In fact, in Bockenheimer's review and analysis and in Koch and Schmidt's report, it seems proved that the microscopic changes in Paget's osteitis deformans and diffuse hyperostosis of bone

Fig. 9.—Phalanges and metacarpals of the upper extremities. Bony changes in the right hand.

of the face and skull are identical. Prince [2] came to the same conclusion, that generalized Paget's disease, showing lesions in the long bones and the skull in middle life, and diffuse hyperostosis of bones of the face and skull, occurring mainly in the earlier decades, are due to the same process as far as gross and microscopic study reveals. He said that if there is mono-involvement in Paget's disease, why not involvement of the skull, which is hyperostosis cranii and, going a step further, why make upper versus lower jaw involvement a criterion of differentiation?

2. Prince, Morton: Tr. A. Am. Phys. **17**:382, 1902.

He expressed the belief that the diseases cannot be differentiated and regarded the condition as a trophic disorder. Wyllie [3] drew attention to neurologic symptoms associated with encroachment on basal neural and vascular foramina including the foramen magnum and reported two cases showing the syndrome of compression of the cord, and one with diplopia. Gregg [4] discussed cases of Paget's disease showing impaired smell, sight, hearing and taste and transient neuralgias due to crowding of nerves by proliferating bony structures.

Fig. 10.—Phalanges and metacarpals of the lower extremities. Bony changes in the right foot.

In 1891, von Recklinghausen [5] described pathologic changes in three cases of generalized disease of the bone, called them fibrous osteitis with

3. Wyllie: Occurrence in Osteitis Deformans of Lesions of the Central Nervous System with Report of Four Cases, Brain **46**:336, 1923.

4. Gregg, D.: Neurologic Symptoms in Osteitis Deformans, Arch. Neurol. & Psychiat. **15**:613 (May) 1926.

5. Von Recklinghausen: Die fibrose oder deformirende ostitis, die Osteomalacie und die osteoplastiche Carcinose in ihren gegen zeitigen Beziehungen, Festschrift R. Virchow, Berlin, 1891, pp. 81.

tumor and cyst formation and correlated and classified pathologic conditions of the bones in which there occur fibrous transformation of the marrow with absorption of old bone, formation of new bone, deposit of cartilage and development of cysts and giant cell sarcoma. He called these all malacias. In his original clinical description and in the large majority of cases reported later, typical cases presented history of pain in a long bone fracture with or without violence and slight or enormous deformity in young adults. In a few cases of long standing, fibrotic changes and formation of cysts were seen in cranial bones, a late and minor involvement. Von Recklinghausen wanted to place the clinical disease in the same category with Paget's osteitis deformans. Elmslie [6] in England, however believed that Paget's disease is so clearly defined clinically that it should be kept apart, in spite of certain pathologic similarities and in spite of the fact that cysts may arise as a minor feature in Paget's disease.

With many subsequent reports of cases, reviews, analyses and reclassifications have been made, with the emphasis mainly on pathology in the German literature; in the English and American literature, in general, an effort has been made to balance things on the clinical side. It seemed feasible to think of this large group of diseases of the bone in two broad categories: the group of osteitis deformans (Paget) and diffuse hyperostosis (leontiasis ossea), and the osteitis fibrosa cystica group. In an attempt to arrive at final diagnosis in this case, it was found impossible to place the case definitely in either group. I hesitate to claim it as unique, but I found no case that showed so unmistakably the progression from the condition described as hyperostosis of the face and skull to a definite picture of osteitis deformans, as far as the appearance of the bones of the skull is concerned, and at the same time an apparent transition in the long bones to an osteitis fibrosa cystica, with thinning and threatening fractures. Clinically and symptomatically, the case does not conform to any one of these diseases in its classic form.

Following another line of investigation, dystrophies of the bones during childhood, one finds the foregoing conditions considered atypical. Of the diseases characteristic of this age period, one can exclude rickets, certain malformations of the face associated with adenoidismus, malformations of the skull alone, as oxycephalus, acrocephalus, scaphocephalus, and those with cranial and facial dystosis associated with symmetrical malformations in the extremities, as exostoses and synostoses about the joints, or with deformity of the clavicle. Crouzon,[7] in 1912, described what he claimed was a new

6. Elmslie: Brit. J. Surg. **2**:17, 1914.

7. Crouzon: Dysostose craniofaciale hereditaire, Bull. et mém. Soc. méd. d. hôp. de Paris, May 10, 1912.

clinical type, which he called hereditary craniofacial dysostosis. He reported the cases of a mother and child in whom similar deformities of the face and skull appeared in the first year of life. These were accompanied by primary optic atrophy, exophthalmos and external strabismus. Whether the last was due to extra-ocular palsy or to displacement of the eyeball was not clear from the text. The deformities became fixed in the first year of life and did not progress; there were no changes in the rest of the skeleton. Crouzon insisted on the condition being hereditary and familial and not congenital. He said that the etiology and pathogenesis were obscure; he called it an hereditary anomaly. Chatelin [8] based his doctor's thesis in 1913 on Crouzon's disease, described the roentgen manifestations and analyzed and compared these with the roentgen observations in other dystrophies of the skull of childhood, confirming the contention that Crouzon's disease is a distinct entity. However, none of these cases showed a condition similar to that described by Dr. Schwartz in this case. Debré [9] reported eleven cases of Crouzon's disease in three generations. Comby,[10] in 1926, reported a case which he identified clinically with Crouzon's cases in every respect except that the condition in his own case was nonhereditary and nonfamilial; in addition to deformities of the skull, the case also presented a deformity of one elbow corresponding to genu valgum in the lower extremity. There is no reference to x-ray studies in Comby's brief report. Although there are suggestive similarities in my case and that of Comby, I do not feel justified in identifying them nor in abandoning the concept of this case as stated earlier in the paper.

Superficially, the case seems to offer little that is of interest from the neurologic point of view. No serious attempt has been made (for purposes of this presentation) to study the case carefully from the point of view of pathologic changes of the bones or clinical orthopedics. However, the disease described here showed multiple lesions in the skeletal system starting in the first year of life which cannot be accounted for on the basis of trauma, bacterial or toxic infection, or new growth. The lesions are confined to the right side except in the skull and, to a minor degree, in the sacrum. It seems justifiable to suggest an interpretation of it as a bone trophic disturbance dependent on hypothetic primary disease of trophic centers in the central nervous system. In the absence of neighboring symptoms and signs, there is no clue here as to localization, and I do not feel that I can support or refute recent work in which tentative localization of such a center is made in the region of the floor of the third ventricle. Further observation and demonstration of pathologic specimens are, of course, essential to definite conclusions.

8. Chatelin: Thése de Paris, 1914.

9. Debré, quoted by Chatelin (footnote 8).

10. Comby: Bull. et mém. Soc. méd. d. hôp. de Paris **50**:1327 (July 23) 1926.

Progress in Pediatrics

DIAPHRAGMATIC HERNIA IN INFANCY AND IN CHILDHOOD *

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AND
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In the last two decades the subject of diaphragmatic hernia has been receiving increasing attention largely as a result of the almost universal routine employment of roentgenologic examination and partly as a result of the fact that a large number of traumatic cases occurred during the World War. The recognition of a case of diaphragmatic hernia in a new-born infant led us to review the literature on the subject and, while we found that several studies had been made on adults (Giffin,[1] Åkerlund,[2] Richards,[3] Quenu,[4] Cade and Montez [5]), we noticed that only one report (Kienbock's [6]) was concerned with infants and children. Kienbock's study, however, was made on a comparatively small number of patients (twenty-two). Latta's [7] study in 1922 was concerned purely with the pathologic examination, and little attention was given to symptomatology and physical signs. We therefore thought it advisable to review the literature from 1912 to the present time and to collect reports of the cases occurring in infants and in children up to 10 years of age, in an attempt to establish the symptomatology of this disease and to advise, if possible, methods of diagnosis. The age incidence, sex, location, size, position of the hernial opening, character of the hernia and the hernial contents were noted (tables 1, 2 and 3). Particular attention was paid to symptomatology, to the number of cases recognized during life and to the methods employed in making the diagnosis. Errors in diagnosis, especially when roentgenograms had been employed, were noted, since roentgenography offers the only certain method of diagnosis during life.

Only the literature subsequent to 1912 was reviewed, because Giffin [1] made a careful study of all cases reported prior to this time and stated

* Submitted for publication, Feb. 1, 1929.

1. Giffin: Ann. Surg. **4**:388, 1912.
2. Åkerlund: Acta radiol. **6**:3, 1926.
3. Richards: Ann. Otol. Rhin. & Laryng. **32**:1145, 1923.
4. Quenu: Étude Clinique et Operatoire, Thèse, Paris, 1920, p. 136.
5. Cade and Montez: Ann de méd. **6**:245 (Sept.) 1919.
6. Kienbock: Fortschr. a. d. Geb. de Roentgenstrahlen **21**:411, 1913-1914.
7. Latta, J. S.: Congenital Deficiency of Diaphragm, Am. J. Dis. Child. **24**:297 (Oct.) 1922.

that only six cases were observed in children under 5 years of age. It may be of interest to add that in only fifteen of his entire series of 650 cases was the condition recognized during life. From 1912 up to the present time, we were able to collect reports of eighty-one cases in children up to 10 years of age; of these twenty-five were recognized during life. Undoubtedly diaphragmatic hernia occurs more frequently than these figures would indicate, because even with the aid of roentgenography there is no question that many cases go undiagnosed. We herewith report an additional case, diagnosed during life and confirmed by necropsy.

REPORT OF CASE

History.—A boy was born on Sept. 28, 1928. The delivery was normal. It was the mother's first pregnancy. The infant was not cyanotic at birth and cried vigorously almost immediately after delivery. Twenty-four hours after delivery it was put to the breast and allowed to nurse; it sucked vigorously, and no unusual symptoms were noted during the first four feedings. At the fifth feeding, that is, on the evening before we examined the infant, it was noticed that it sucked the breast vigorously for two or three minutes, stopped, appeared to be in great pain

TABLE 1.—*Age Groups of Cases Studied*

Age Groups	Number of Cases
Stillborn	13
1 to 24 hours	30
2 to 18 days	6
1 to 12 months	15
1 to 8 years	18

and immediately afterward regurgitated the milk through the mouth and nose. Cyanosis made its appearance at the same time but soon disappeared. At our first examination on September 30, the infant was given a bottle of water, and the same train of symptoms manifested themselves. In addition, it was observed that the regurgitation through the nose and mouth was not forcible in character but rather that the fluid trickled out. It was also noticed that the dyspnea and cyanosis disappeared as soon as the infant was held in an erect position. Examination of the chest disclosed some displacement of the apex beat to the left; on percussion, a tympanitic note was heard on the right side, posteriorly, at the seventh interspace, about 1 inch (2.5 cm.) from the vertebral column. The breath sounds were distant over this area, but a splashing sound could be heard. The abdomen was not retracted, and no masses could be felt. The fontanel was depressed. The mucous membrane of the mouth was red and dry. Otherwise, the physical examination gave negative results. Since we observed these signs in a case of eventration of the right side of the diaphragm with nonrotation of the stomach, it naturally occurred to us that we were dealing with a similar condition here. A catheter was passed through the nose into the stomach; fluoroscopic examination showed the catheter in the midline, until just above the diaphragm, at which point it passed over to the right, forming an obtuse angle; it was again seen on the right side above the diaphragm and liver. A suspension of barium in water was injected through the catheter. The stomach filled up rapidly, but after 10 cc. was injected it was noticed that the esophagus became widely dilated and reverse peristalsis occurred. At the same time, part of the barium suspension was regurgitated through the mouth and nose. Enough

barium remained in the stomach, however, to show that the organ was definitely in the right side of the thorax. After a few minutes active peristalsis could be observed in the stomach, but no barium could be seen leaving it. After forty-five minutes, practically no barium was seen in the small intestine. Several plates were taken in the dorsoventral, ventrodorsal and both lateral positions. The roentgenograms were interpreted as follows:

In the plate taken before the tube was inserted into the esophagus (fig. 1), there was some ill defined mottling in the right paracardiac region. No other changes were noticed. The cardiac and vascular shadows were normal; the trachea was

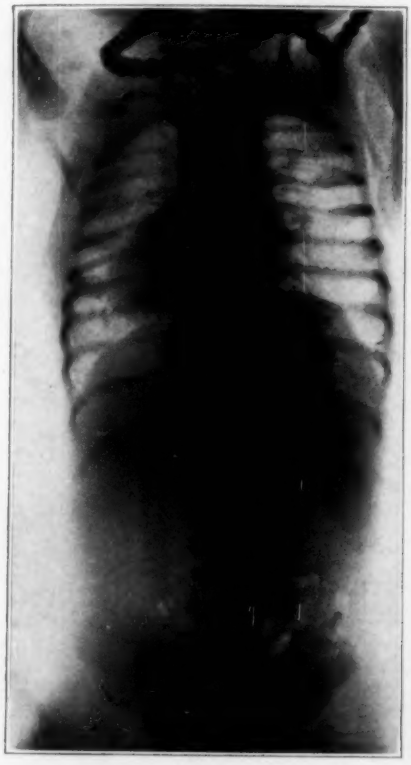

Figure 1

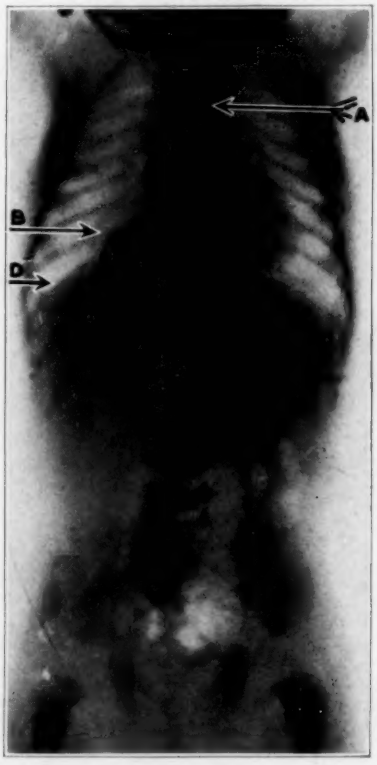

Figure 2

Fig. 1.—Anteroposterior view before the introduction of the barium mixture.

Fig. 2.—Anteroposterior view after the introduction of barium mixture (catheter in situ). *A*, catheter; *B*, roughly circular portion of stomach (supradiaphragmatic), and *D*, diaphragmatic leaflet.

not deviated. The right leaflet was at the level of the ninth rib posteriorly, the left at a slightly lower level. No defects were seen in either leaflet. The liver and spleen were in their normal positions.

A catheter was inserted into the esophagus and a barium mixture injected (fig. 2). The esophagus, as outlined by the catheter, was straight, not tortuous. It lay slightly anterior to the thoracic spine and at about the sixth dorsal vertebra;

it was somewhat to the left of the midline; below this point, it swerved and crossed slightly to the right of the median line to terminate at the level of the eighth dorsal vertebra in an irregularly circular cavity that contained barium, undoubtedly a portion of the stomach. This portion of the stomach lay above the diaphragmatic leaflet directly anterior to the spine, the greater portion being to the right of the midline, 1½ inches (3.7 cm.) transversely, 1¼ inches (3.1 cm.) vertically and an inch anteroposteriorly. From the anterior portion, at a point just above the lower border of the roughly circular portion of the stomach, a narrow tubular portion of the stomach could be seen which passed downward, forward and

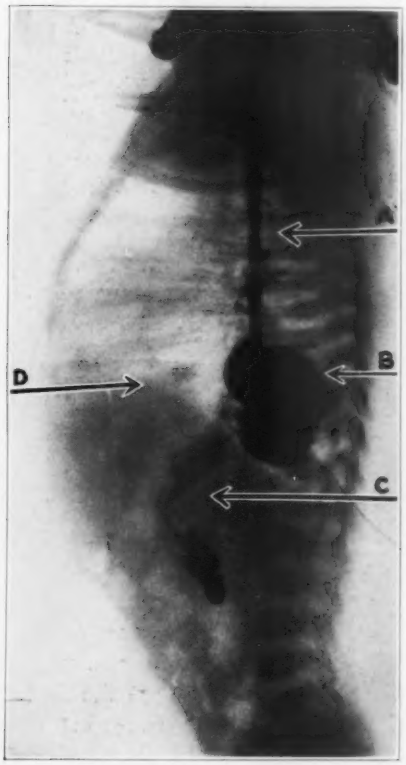

Figure 3

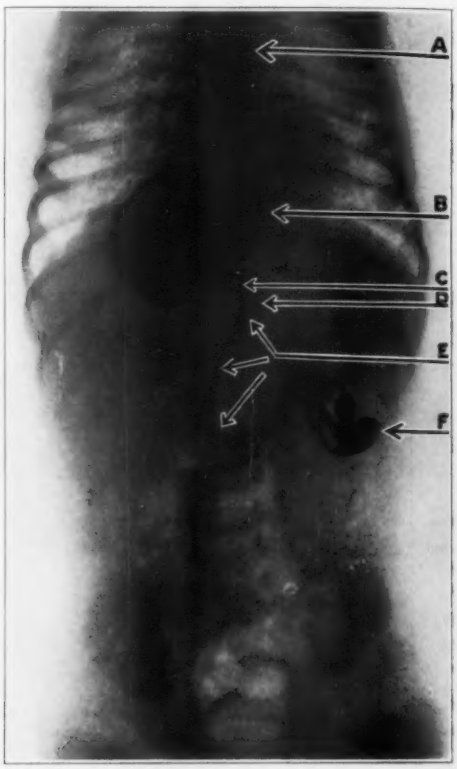

Figure 4

Fig. 3.—Lateral view after the introduction of the barium mixture (catheter in situ). *A*, catheter; *B*, roughly circular portion of stomach (supradiaphragmatic); *C*, tubular portion of stomach (infradiaphragmatic), and *D*, diaphragmatic leaflet.

Fig. 4.—Anteroposterior view after the introduction of barium mixture (catheter withdrawn). *A*, esophagus; *B*, roughly circular portion of stomach (supradiaphragmatic); *C*, tubular portion of stomach (infradiaphragmatic); *D*, pyloric valve; *E*, duodenum, and *F*, small intestines.

to the left through the dome of the diaphragm at a point corresponding roughly with the normal esophageal opening. The tubular portion was about 1 inch in length and terminated at the pylorus which lay at the level of the twelfth dorsal

vertebra. The duodenum was plainly visualized and was normal, except that the descending portion was somewhat elongated and apparently overlay the spine. The colon, as outlined by gas, appeared to occupy normal relations.

After three hours, there was considerable retention in the supradiaphragmatic portion of the stomach (fig. 5).

After twenty-four hours a speck of barium was still visible in the stomach; at this time, there was almost complete evacuation of the meal (fig. 6).

A diagnosis of congenital diaphragmatic hernia of the right side was then made.

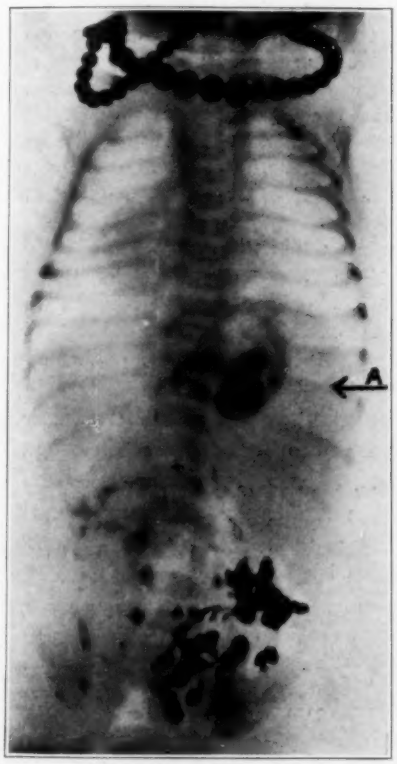

Figure 5

Figure 6

Fig. 5.—Anteroposterior view three hours after the administration of the barium mixture. *A*, retention in the supradiaphragmatic portion of the stomach.

Fig. 6.—Anteroposterior view twenty-four hours after the administration of the barium mixture.

It was apparent from the fluoroscopic examination that the motility of the stomach was only slightly interfered with, but that there was definite interference with the emptying of the organ; it was also noted that quantities of more than 10 cc. were immediately regurgitated. It was therefore thought advisable to feed the infant small quantities (10 cc.) of breast milk every half hour. Regurgitation persisted, however; the cyanosis became more marked; severe dyspnea made its appearance, and on the next day (October 1) evidences of diffuse bronchopneu-

monia were noticed. The infant's general condition grew rapidly worse, and it died on the following day.

Necropsy (Dr. M. A. Goldzieher).—The body was poorly developed and decidedly undernourished.

After the thoracic cavity was opened, a bulging mass was found on the right side, standing out from the diaphragm. This mass was about the size of a hazelnut. It was extrapleural and disappeared on pressure. Yet pressure on the abdomen made the mass reappear.

When the abdomen was opened, all the viscera were found in their usual places with the exception of the stomach, which seemed to disappear behind the liver. After the latter was lifted, it was found that the esophageal opening of the diaphragm was greatly distended, so much so that it admitted the tip of a finger. Through this opening a peritoneal pocket bulged out, which received the stomach. This was the same pocket that could be visualized in the right thoracic cavity.

Both lungs were free and contained air throughout, with the exception of a few scattered areas in the left lower lobe. These solid foci were grayish red and quite friable. On section, a large amount of grayish stringy exudate oozed out from the bronchi.

The heart was well contracted. Its valves were thin and smooth.

The abdominal organs did not show any pathologic changes.

Diagnosis.—The diagnosis was: true diaphragmatic hernia containing the stomach, diffuse bronchitis and broncheolitis, and bronchopneumonia.

COMMENT

Hernia is generally classified as congenital, acquired and traumatic. Because all cases that are not traumatic are probably congenital in origin, we suggest the employment of only two terms in the classification, namely, traumatic and nontraumatic. In the traumatic group are all cases in which there is a history of injury to the diaphragm; in the nontraumatic group are all cases in which no such history is obtained. It is therefore obvious that in all cases in group 1 the condition must be acquired and that in all cases in group 2 it must be congenital; the latter group includes those cases in which the hernia may not be present at birth but in which a congenital weakness of the diaphragm is present, the hernia manifesting itself later in life.

Anatomically, hernia is classified as true when a hernial sac is present and as false, when no hernial sac exists. The most important factor concerned in the production of false hernias is interference with the normal closure of the pleuro-peritoneal membrane during fetal life, resulting in a defect without a sac. True hernias are due to an abnormal development or formation of the diaphragm as a result of local pathologic interference with circulation, or faulty innervation. As in false hernias, these changes occur during fetal life.

In true hernia, the sac may be small or it may be tremendous. In fact, it may be so large as to be mistaken roentgenologically for eventration (Schoenfeld's case [8]). False hernias are much more common than

8. Schoenfeld: Klin. Wchnschr. **5**:1657 (Sept.) 1926.

true hernias. Thus, of the 635 case reports collected by Eppinger,[9] in 561 the hernias were false and in seventy-one, true. Grosser and Lipmann [10] stated that false hernia is from five to six times as frequent as true hernia. Latta [7] also stated that false hernias are much more common than true ones. He collected reports of 127 cases of false hernias, but he made no mention of the number of true ones encountered. Richards [3] reported forty-nine true ones and sixty-seven false ones. In the series of cases reviewed here, there are fourteen true and thirty-six false hernias, leaving thirty-two cases undetermined; of the cases recorded, in 72 per cent the hernias were false and in 28 per cent true (table 2).

As a rule, true hernias are relatively more frequent on the right side than false hernias. In 561 false hernias reported by Eppinger,[9] thirty-four occurred on the right side and 527 on the left; of the seventy-four cases of true hernias, twenty-one were on the right side and fifty-three on the left. Latta,[7] too, in a report of 127 cases of false hernia found that 110 were left-sided. In the case reports collected here, it was noted

TABLE 2.—*Frequency of Location and Type of Hernia*

Side	Number of Cases	True	False	True and False	Type Undetermined
Left	61 (77.2%)	10	24	..	27
Right	16 (20.2%)	3	10	..	3
Right and left	2 (2.6%)	..	1	1	..
Undetermined	3	1	1	..	1
Total	82	14 (28%)	36 (72%)	1	31

that of the thirty-six false hernias, twenty-four were on the left side, ten on the right, one was bilateral and one was undetermined; of the fourteen true hernias, three were on the right side, ten on the left and the position of one was unstated. We are at a loss to explain this discrepancy in the percentages, since it is questionable whether the difference in the age incidence of the two series is a factor.

In false hernias the defect may be small or half of the diaphragm may be entirely absent, giving wide communication between the abdominal and thoracic cavities. In six cases, the left leaflet was entirely absent. Herniation occurs (1) through the esophageal opening, (2) through the foramen of Morgagni or Larry's space, and (3) through the foramen of Bochdelaki. The foramen of Morgagni is found at the junction of the sternal and costal portions of the diaphragm and is really a gap in the musculature which is covered only by pleura and peritoneum. The foramen of Bochdelaki is also a gap in the musculature covered by

9. Eppinger: Allgemeine und Spezielle Pathologie der Zwerchfells, suppl. zur Spezielle Pathologie und Therapie, 1911.

10. Grosser and Lipmann: Arch. f. Gynäk. **68**:780, 1903; Wien. klin. Wchnschr., 1899, p. 657.

pleura and peritoneum and is found posteriorly at the junction of the lumbar and costal portions of the diaphragm. There is quite a difference of opinion as to the frequency with which herniation through the esophageal opening occurs. Thus, Aschoff [11] considered that herniation through the esophageal opening occurs less frequently than through the foramen of Bochdelaki or the foramen of Morgagni. Eppinger [9] found it eleven times in 635 cases. Carmen and Fineman [12] found it but once in seventeen cases. Keith [13] collected reports of thirty-four cases, in only one of which the herniation was through the esophageal opening. Quenu [4] collected reports of 116 cases, and in only two of these did herniation occur through the esophageal opening. Åkerlund,[2] on the contrary, was able to collect reports of twenty-four cases in which the herniation occurred through the esophageal opening. In addition, she stated in her report that esophageal hernia was from six to seven times as frequent as other authors reported it to be. Hume [14] found twelve instances of herniation through the esophageal opening in a series of thirty-five cases. Pancoast [15] found twelve instances of herniation through the esophageal opening in fifteen nontraumatic cases.

Hedblom,[16] too, found twenty-four esophageal hernias in sixty-two nontraumatic cases (38 per cent). In fact, this situation is regarded by many surgeons as the most common site for diaphragmatic hernia. Richards [3] explained this apparent discrepancy by stating that in the living adult, as seen at operation, this situation doubtless is the most common site, but that in the sum total of diaphragmatic hernias the esophageal ones are extremely uncommon. Richards pointed out in addition that, strictly speaking, there is no esophageal opening, i.e., the digestive tract is everywhere intimately enclosed by the dorsal, and above the diaphragm by the ventral mesentery through which the esophagus passes. The only "opening" is that created by the failure of the diaphragmatic muscle to surround the esophagus closely. The more the muscle fails to do this, the greater is the amount of the membranous tissue composed of peritoneum, pleura and interstitial tissue, which, under certain conditions, may become the site of a hernia. For this reason esophageal hernias are commonly of the true type, contained in a sac formed of relapsed membranous diaphragm, intimately attached to the esophagus itself. Indeed, this was exactly the situation in our case. As may be noticed from the necropsy report, the esophagus was not

11. Aschoff: Path. Anat. **2**:873, 1921.

12. Carmen and Fineman: Radiology **3**:26 (July) 1924.

13. Keith: Brit. M. J. **2**:1297, 1910.

14. Hume: Brit. J. Surg. **10**:207 (Oct.) 1922.

15. Pancoast, H. K., and Boles, R. S.: Nontraumatic Left Diaphragmatic Hernia, Arch. Int. Med. **38**:633 (Nov.) 1926.

16. Hedblom, C. A.: Diaphragmatic Hernia, J. A. M. A. **85**:947 (Sept. 26) 1925.

fixed in the diaphragmatic opening, but was so freely movable that angulation actually occurred. In this series of eighty-two cases, eleven hernias occurred through the esophageal opening (20 per cent of the cases in which the location was recorded). When herniation through the esophageal opening occurs, it may be found with equal frequency on either side. In eleven cases of hernia through the esophageal opening, Eppinger [9] found six on the left and five on the right. In this series of eleven hernias through the esophageal opening, six were on the left, three on the right and in two cases no mention was made of the side. Whether the hernia occurs through the esophageal opening or not is of more than academic interest, since in five of the cases collected in which operation was successfully performed, four hernias were through the esophageal opening. The location of the hernia is of decided interest to the surgeon from the standpoint of repair. It will be noticed that twenty-nine hernias were located posteriorly, twenty-four of which were on the left side; eleven were through the esophageal opening, seven were central, in six there was a complete absence of the diaphragm—all on the left

TABLE 3.—*Position of Hernia*

Position	Number of Cases
Posterior	29 (52.7%)
Esophageal	11 (20%)
Central	7 (12.8%)
Complete absence of one leaflet	6 (10.9%)
Anterior	2 (3.6%)
Unstated	27

side—and finally, two were located anteriorly; twenty-seven cases were undetermined. Of the cases in which the location of the hernia was recorded, 52.7 per cent were posterior, 20 per cent esophageal and 12.8 per cent central; 10 per cent showed complete absence of one leaflet and 3.6 per cent were anterior (table 3).

Other weak points in the diaphragm through which herniation may occur are the openings for the passage of the aorta, vena cava and smaller vessels and nerves. However, we have been unable to find a single case in which hernia occurred through the aortic opening. This may probably be due to the fact that the aortic opening is anatomically and developmentally not in the diaphragm but behind it.

The majority of hernias occur on the left side. In the cases reviewed, sixty-one, or 77.2 per cent, were on the left side; twelve, or 20.2 per cent, were on the right side, and two, or 2.6 per cent, were on both sides. Three cases were undetermined. This is unquestionably due to the protection afforded by the liver, although it has been shown that at the time of the development of the diaphragm in the embryo the left half of the liver affords the left leaflet as much protection as it does the right. The liver, however, does decrease the possibility of misplacement of viscera into the right pleural cavity. That this is not always true, however, may be

seen from the report of Mitchell's case [17] in which the hernial opening was on the left side, but the transverse colon, omentum and stomach were found in the right thoracic cavity. In our case, too, although the hernial opening was on the left side, the stomach was found in the right thoracic cavity.

The degree of herniation is no criterion of the extent of the defect in the diaphragm. Thus, McCleave [18] reported a case in which almost all of the intestines were herniated through an oblique slit about 1 inch long on the left side, close to the point of penetration of the great vessels and the esophagus.

The organs which may become herniated are, in the order of their frequency, (1) the stomach, (2) the transverse colon, (3) the omentum, (4) the small intestine, (5) the liver, (6) the pancreas, and (7) the kidney (Lichtenstern [19]).

Sex may be a factor; Hedblom [16] found that 78.6 per cent of 197 cases were in males and 24.4 per cent in females. In this series twenty-eight, or 68.9 per cent, were in males, and thirteen, or 31.1 per cent, in females; in forty-one cases, the sex was not recorded. While this preponderance of diaphragmatic hernia in men may be explained by occupation, mode of living, etc., this explanation does not hold true for infants and children. We are at a loss, however, to explain the occurrence of more than twice as many cases in males as in females.

Pneumonia was diagnosed or present in a fairly large number of cases in this series. Whether the collapsed lung on the same side or the compensatory hypertrophy of the lung on the opposite side offers a fertile field for infection is questionable. It is quite possible that in some cases a mistaken diagnosis of pneumonia was made because of the presence of dulness or flatness on percussion or tubular breathing on auscultation. On the other hand, bronchopneumonia due to aspiration is not an uncommon terminal event.

SYMPTOMS

Attempts have been made by Cade and Montez [5] to divide hernias clinically into groups corresponding to symptoms and signs referable to the gastro-intestinal, cardiac and respiratory systems, depending on the predominance of one group of symptoms. Predominating system symptom complexes do exist in infants and children but are dependent to a great extent on the age of the patient, and it therefore seemed more advisable to us to study the semeiology of this disease from the point of view of the patient's age. We

17. Mitchell: Southern M. J. **10**:561 (July) 1917.

18. McCleave, T. C.: Diaphragmatic Hernia, Am. J. Dis. Child. **13**:252 (March) 1917.

19. Lichtenstern: Berl. klin. Wchnschr., 1874, p. 497.

have therefore divided the symptoms of diaphragmatic hernia into three groups, depending on the age of the patient: first, the symptoms in infants who lived twenty-four hours or less; second, those of infants who lived from one day to the end of the new-born period, and, third, those of infants and children.

In new-born infants the outstanding symptom, cyanosis, was present in nineteen cases (table 4). Cyanosis is due to circulatory disturbances because of pressure on the heart and larger vessels produced by the herniated organs and to mechanical interference with pulmonary action. It may be constant or may come only in attacks occurring especially after the feeding. At times the cyanosis disappears when the patient is held in the upright position. In Davis' [20] patient, as well as in ours, cyanosis was present only after feeding and disappeared when the infant was held in the upright position.

TABLE 4.—*Symptoms and Signs in Order of Frequency from Birth to First Twenty-Four Hours of Life (Thirty Cases)*

Symptoms	No. of Cases	Signs	No. of Cases
Cyanosis	19	Dextrocardia	6
Peculiar single weak cry	8	Heart displaced to left	2
Feeble attempts at respiration	8	Tympany on percussion of involved side of chest	2
Failure to breathe with heart beat present	8	Absent breath sounds	2
Dyspnea	5	Gurgling sounds heard in chest	1
Difficulty in nursing	3	Dulness on percussion of involved side of chest	1
Convulsive attempts at respiration	2	Bulging thorax	1
Cough	1	Retraction of abdomen	1
		Distention of abdomen	1

Next in importance to cyanosis is some disturbance in the respiratory apparatus. The infant may fail to breathe spontaneously in the presence of a strong heart beat. This was true of eight new-born infants in the series of case reports that we collected. Feeble attempts at respiration were present in eight cases, while dyspnea was present in five cases. At times the infant may also make convulsive attempts at respiration, breathing with marked effort and bringing all the auxiliary muscles of respiration into play. These convulsive attempts at respiration were present in two cases. Another important symptom and one which Guenoit [21] considered characteristic of diaphragmatic hernia in the new-born is a peculiar single, weak cry emitted by the infant immediately after delivery; this was present in eight cases. Various other observers ascribe much importance to this cry. Indeed, Schreiber [22] suspected hernia in his patient because of this peculiar cry, unexplained cyanosis

20. Davis, C. M.: Diaphragmatic Hernia in New-Born, Am. J. Dis. Child. **24**:356 (Oct.) 1922.

21. Guenoit, quoted by Prat: J. de chir. **16**:43, 1920.

22. Schreiber: Arch. de méd. d. enf. **25**:607 (Oct.) 1922.

and dextrocardia. Von Dongen [23] also noticed this cry coupled with the absence of attempts at respiration in his patient. The infant became intensely cyanotic following the initial cry. Quenu [24] stated that cyanosis and death following this initial weak, peculiar cry are particularly characteristic of this condition. That this does not always hold true is seen by the fact that many infants cry lustily following delivery. This happened in Rüder's [25] case. His patient cried lustily following delivery but went into a state of collapse immediately after the cord had been severed, and died within a few minutes. Our patient also cried lustily at birth and presented no symptoms until about twenty-four hours after delivery.

The infrequent occurrence of dyspeptic symptoms during the first twenty-four hours of life can readily be explained. In only three of this series of cases was there difficulty in nursing, and in only one was there vomiting after a feeding. This may be attributed to the fact that new-born infants with extensive hernias do not live long enough to nurse. Of thirty new-born infants, thirteen lived less than one hour and nine lived less than twenty-four hours, death ensuing shortly or directly after birth.

Cough was present in one case, that of Griewe; [26] it occurred in an infant directly after birth and persisted. Griewe considered the cough an etiologic factor in the production of hernia in his case.

In summarizing the symptoms, as they occur in new-born infants, one may say that cyanosis coupled with some respiratory difficulties seems to be the outstanding feature. The respiratory difficulty may vary from a total absence of respiration, so that the child is stillborn, to weak, feeble attempts at respiration; or, on the other hand, dyspnea or convulsive attempts at respiration may be present. In addition, the peculiar, single weak cry already described is of extreme value in establishing a diagnosis of hernia. In view of the fact that there were thirteen stillborn infants whose death was due directly to the presence of diaphragmatic hernia, it seems to us that one should at least suspect this condition as a possible cause of stillbirth.

In those patients in whom the respiratory and circulatory symptoms are marked, the hernia is usually extensive and is incompatible with life. That diaphragmatic hernia is not always incompatible with life may be seen from the number of nontraumatic cases reported in patients ranging in age from a few days to adults well advanced in life.

The second age group (table 5) presented symptoms related mainly to the cardiac and respiratory systems, as does group 1, except that dyspeptic symptoms were relatively more frequent and physical signs were

23. Von Dongen: Nederl. Tijdschr. v. Geneesk. **1**:582, 1922.
24. Quenu: Arch. méd. chir. de l'app. respir. **1**:356, 1926.
25. Rüder: Deutsche med. Wchnschr. **43**:160, 1917.
26. Griewe: Arch. Pediat. **37**:593 (Oct.) 1920.

more often elicited than in group 1. More definite conclusions cannot be drawn because of the paucity of cases in this age group, only six cases being reported.

In group 3 (table 6), which consists of infants over 1 month of age and older children, vague dyspeptic symptoms, not in proportion to the gravity of the lesion, are as a rule, present.

TABLE 5.—*Symptoms and Signs in Order of Frequency in Infants Whose Ages Ranged from One Day to One Month (Six Cases)*

Symptoms	No. of Cases	Signs	No. of Cases
Cyanosis	5	Dextrocardia	3
Dyspnea	2	Absent breath sounds on involved side of chest	2
Difficulty in feeding	1	Tympany on percussion of involved side of chest	1
Vomiting	1	Bulging thorax	1
Rapid, irregular breathing	1	Retraction of abdomen	1
Cough	1		
Loss of weight	1		

TABLE 6.—*Symptoms and Signs in Order of Frequency in Children One Month of Age Up to Ten Years of Age (Thirty-Three Cases)*

Symptoms	No. of Cases	Signs	No. of Cases
Vomiting	16	Tympany on percussion of side of chest involved	11
Cyanosis	12	Dextrocardia	7
Dyspnea	9	Dulness to flatness on percussion of side of chest involved	7
Constipation	9	Diminished breath sounds on side of chest involved	6
Failure to gain in weight	8	Rales	6
Epigastric pain	7	Unilateral bulging of chest	6
Cough	7	Diminished excursion of side of chest involved	4
Diarrhea	4	Retraction of abdomen	4
Inability to take food	3	Temperature	4
Discomfort after meals	3	Tinkling sounds heard in chest	3
Acute onset with symptoms of obstruction	3	Bronchial breathing	3
Anorexia	2	Absent breath and voice sounds on side of chest involved	2
Weakness	2	Gurgling sounds heard in abdomen	2
Short attacks of coma	1	Mass in epigastrium	2
Hematemesis	1	Distention of abdomen	2
		Rapid heart beat	2
		Pallor	2
		Symmetrical bulging of thorax	1

The symptoms depend on the organ herniated. If the stomach was herniated and the cardiac or pyloric portions were partly occluded because of the pathologic position of the organ, vomiting was the most prominent symptom, occurring in sixteen cases. This can be well correlated with the pathologic observations, namely, that the stomach is the organ most frequently herniated. The vomiting is not projectile in type, and it usually has a definite relation to meals, occurring after a feeding. The child usually complains of a sense of fulness or oppression in the epigastrium after meals, which is relieved by emesis. Attacks of vomiting may be accompanied by epigastric pain, the symptoms occurring in crises; i.e., there are times when the patient is entirely free from symptoms,

while at other times the pain is severe. These crises are due to a partial occlusion of the herniated viscus. Death usually occurs in these cases from strangulation. Vomiting is a source of danger, not only, because of the inanition which may result, but also because of the possibility of the development of bronchopneumonia from aspiration of the vomitus. This is especially apt to occur in young infants. In fact, the immediate cause of death in our patient was aspiration pneumonia.

Constipation, failure to gain in weight, anorexia, inability to take food and discomfort after meals are the other dyspeptic symptoms usually present. Constipation was present in nine cases, failure to gain in weight in eight cases, epigastric pain in seven cases, diarrhea in four cases, inability to take food in three cases and anorexia in two cases. Difficulty in feeding or inability to take food is usually due to pressure on the esophagus or displacement and angulation of the esophagus. In three cases the onset was acute, with symptoms of intestinal obstruction, i.e., vomiting, absolute constipation, epigastric pain and collapse.

Hematemesis was present in only one case, in a child, aged 3½ years. Hematemesis points to the presence of gastric ulcer, which is not an infrequent occurrence in adults with diaphragmatic hernia. It is probably due to an interference with the circulation (Plenk [27]).

While dyspeptic symptoms are more common in older children, cardiorespiratory symptoms are by no means infrequent. Thus, cyanosis, the outstanding symptom in new-born infants, was present in twelve cases in group 3. The same factors operate in producing the cyanosis here as in the case of infants. The type of respiratory symptoms encountered here differs from that in the new-born in that dyspnea was the more common complaint, having been present in nine cases. Cough was present in seven cases and was probably due to the disturbance in the respiratory mechanism caused by the herniated organs. Weakness was the chief complaint in two cases. Short attacks of coma were present in two cases, in children 2 months and 9 months of age, respectively; in addition, attacks of cyanosis and dyspnea were noted.

Thus it can be seen that the symptoms of diaphragmatic hernia in older children differ from those in the new-born infant in that dyspeptic symptoms are the more common and constant complaints in older children. It can be further noted that respiratory symptoms occur and are coupled with the dyspeptic symptoms.

Physical signs can usually be elicited in every case, but they necessarily depend on several factors, such as the age of the patient, the organ herniated, the degree of herniation, and the relation of food intake to the time of examination. Changes in the position of the patient may also at times influence the results of the physical examination.

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27. Plenk: Wien. klin. Wchnschr. **35**:339 (April) 1922.

In new-born infants the outstanding physical sign was dextrocardia, which was present in six cases. In the six cases in the second group it was present three times, while in children over 1 month of age and up to 10 years of age, it was present seven times. In four cases of the entire series a clinical diagnosis of dextrocardia was made. Indeed, in 1915, Grulee [28] stated that a clinical diagnosis of dextrocardia in a new-born child always led him to suspect the presence of a diaphragmatic hernia. In two cases in new-born infants, the heart was displaced to the left. In a comparison of the tables of physical signs in new-born infants and in older children, it will be seen that there exists a relative paucity of signs in new-born infants. This may be due to the fact that the infants do not live long enough to permit a complete examination or that careful search is not made for these chest signs. In thirty new-born infants there were only two who showed tympany on percussion of the side involved. An absence of breath sounds was noted in two cases, dulness on percussion of the involved side of the chest in one case, bulging of the thorax in one case, gurgling sounds in the chest in one case, retraction of the abdomen in one case, and distention of the abdomen in one case. These are the signs which one should look for in suspected cases of diaphragmatic hernia in new-born infants.

In older children, physical signs can usually be elicited in every case. The outstanding physical signs are those elicited by an examination of the chest and are referable in the majority of cases to the respiratory system. On inspection, some asymmetry of the chest may be observed. In six cases there was a unilateral bulging of the chest wall. In two young infants, a symmetrical bulging of the thorax was noticed. In older children in whom the hernia has existed for some time, disturbed relationships in intrathoracic pressure interfere with the normal symmetrical development of the chest, thus producing asymmetry. On further inspection a diminished excursion of the chest wall on the side affected may be noted; this is due to failure of the lung to expand on the side involved. The heart may be displaced to one side or the other, depending on the location of the hernia, and dextrocardia is usually present, as was previously noted. On palpation, tactile fremitus was absent on the affected side. It is by percussion that the most interesting observations are made; the type of note elicited, however, will depend on the organ herniated. If the liver is herniated, there is flatness of the right side posteriorly from the third or fourth rib downward; if the stomach or the intestines are herniated, percussion downward from the third or fourth rib anteriorly and from the sixth or seventh rib posteriorly gives a tympanitic note. If the stomach is full, however, the tympanitic note may be replaced by flatness which is shifting in character. A note ranging from dulness to flatness on the affected side was

28. Grulee: Internat. Clin. **25**:100, 1915.

present in seven cases, and tympany was present in eleven cases in the older children and in three cases in the younger group, making a total of fourteen cases. Thus tympany noted on percussion is one of the outstanding observations and in conjunction with dextrocardia strongly points to the presence of a diaphragmatic hernia. On auscultation, the breath sounds are usually diminished or absent; in six cases the breath sounds were diminished, and in four cases they were entirely absent. In three cases tubular breathing was heard, while moist râles were heard in six cases. Distant amphoric breathing with splashing, tinkling sounds and whispered pectoriloquy may be heard and may thus lead to an incorrect diagnosis of hydropneumothorax. Tinkling sounds heard in the chest were described in three cases. In eliciting the chest signs, it is advisable to examine the child before and directly after a feeding in order to note whether or not there is any change in the chest. This is of value in establishing the diagnosis.

As one would expect, physical symptoms in the abdomen are far less common than in the chest. Retraction of the abdomen was noticed in six cases, four of which were in older children. Retraction of the abdomen in the presence of unusual chest signs should lead one at least to suspect the presence of a diaphragmatic hernia. Indeed, Foster [29] diagnosed a left-sided diaphragmatic hernia without the aid of roentgenograms, because of the presence of these signs. Distention of the abdomen was present in four cases, two of these being in older children. Distention is usually due to complete or partial strangulation of the herniated viscus. An interesting condition in two cases was a mass in the epigastrium, which on operation proved to be distended stomach caused by pyloric obstruction.

The presence of a diaphragmatic hernia can only be suspected from the physical signs and symptoms; roentgenologic studies are essential to establish the diagnosis. Fluoroscopic examination may be of value and will, at times, reveal paradoxic movements of the diaphragm.

It must be emphasized, however, that negative roentgenologic examinations do not rule out the presence of a diaphragmatic hernia. This is especially true when the hernia is small. Soresi [30] reported a case in which only a small portion of the lesser curvature of the stomach was herniated through a gap in the diaphragm, 3 cm. in length, near the esophagus. In this case repeated roentgenograms and test meals failed to reveal any abnormalities of the gastro-intestinal tract, except a delay in the emptying time of the stomach. The diagnosis was made at operation.

Another source of error is improper interpretation of the roentgenograms. This occurred in ten cases in this series. Two cases were interpreted as eventration (Schoenfeld,[8] Kienbock [6]), one case as

29. Foster: New York M. J. **112**:77 (July) 1920.
30. Soresi: Ann. Surg. **69**:254 (March) 1919.

esophageal diverticulum (Pauchet and Luguet [31]); one case as a tuberculous process in the lung (Southby [32]); in two cases no interpretation of the plates could be made (Schafer,[33] DeBuys [34]); three cases were diagnosed as pneumothorax or pyopneumothorax (Hume,[14] Fidler,[35] Olmstead [36]) and one was interpreted as pneumonitis (Guistinian [37]).

Difficulties in the roentgenologic diagnosis of diaphragmatic hernia may be due to any of the following reasons: (1) Spontaneous reduction of the herniated organ may take place at the time the roentgenogram is made. (2) The opaque mediums may not pass through into the herniated organs because of strangulation. (3) Herniation of a solid viscus such as omentum, liver or spleen, which cannot be visualized by barium, may be present.

The most frequent complication is strangulation of the herniated viscus. Strangulation is characterized by sudden sharp pain in the epigastrium, usually after a feeding; this is soon followed by signs of shock. The breathing is rapid and costal in type; the pulse is rapid and small in volume; the temperature is subnormal, as a rule, and restlessness is marked. Vomiting is a prominent symptom, and the vomitus frequently contains blood. The abdomen is retracted, and unusual chest signs are found. Death may follow at once or, if the patient survives the initial shock and the strangulation is not relieved immediately by operation, gangrene and perforation of the strangulated viscus occur with a resulting pyopneumothorax and sepsis. This complication occurs in about 15 per cent of the cases and is due either to torsion, ingestion of large amounts of food, increased intra-abdominal pressure, straining at stool or falls on the abdomen; or it may be due to the formation of adhesions which interfere with peristalsis. Strangulation may occur even when the hernial opening is large; and it may be the first evidence of a diaphragmatic hernia. The sudden onset of unusually severe signs and symptoms of intestinal obstruction coupled with unusually severe chest signs should always lead one to suspect the presence of a diaphragmatic hernia. A complication which occurs more often in adults than in children but which may at times be found in older children is ulceration of the stomach.

Another complication which is less frequent is the occurrence of a purulent effusion either from rupture of a gastric ulcer into the chest cavity or as a result of injury to the herniated organ from thoracentesis.

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31. Pauchet and Luguet: Bull. Acad. de méd., Paris, vol. 92, p. 92, 1928.
32. Southby: M. J. Australia **1**:189 (Feb.) 1924.
33. Schafer: Tr. Chicago Path. Soc., 1917-1918, p. 318.
34. DeBuys, L. R.: Anomaly of Diaphragm with Herniation into Thorax of Certain Viscera, Am. J. Dis. Child. **19**:55 (Jan.) 1920.
35. Fidler: Berl. klin. Wchnschr. **11**:1795, 1914.
36. Olmstead: Northwest. Med. **27**:169 (April) 1928.
37. Guistinian: Semana méd. **1**:314 (Feb.) 1927.

DIAGNOSIS

The clinical diagnosis of diaphragmatic hernia is difficult, because of the variety of symptoms which this condition presents. In the series of eighty-two cases studied, an intravitam diagnosis was made in twenty-seven cases, but only six of these were diagnosed clinically; twenty-one were diagnosed by routine roentgenologic studies, hernia not being suspected. Forty-seven of the cases were diagnosed at autopsy. Four were diagnosed at operation, and in four it was not stated how the diagnosis was made. Schreiber [22] diagnosed a case of left-sided, false diaphragmatic hernia clinically in a new-born infant because of the single, weak, peculiar cry present immediately after delivery, unexplained cyanosis and dextrocardia—three cardinal manifestations of hernia in new-born infants. An interesting feature of his case was the complete absence of the left leaflet of the diaphragm. The diagnosis was confirmed by roentgenograms and necropsy. Davis [29] diagnosed a case clinically in an infant, aged 8 days, because of attacks of cyanosis and dyspnea which were relieved by holding the child in an upright position. The cyanosis became intense after feedings. In addition, there was bulging of the thorax, retraction of the abdomen, a tympanitic note on percussion of the side of the chest involved, and absent breath sounds. The presence of the unusual chest signs helped confirm the diagnosis. The diagnosis in this case was confirmed by roentgen studies. The child lived until 4 months of age, thriving and developing normally until signs of strangulation made their appearance, resulting in death. The third case diagnosed clinically was that of Clark,[38] in a new-born infant who presented the following symptoms: cyanosis, a peculiar weak moaning cry, dyspnea and difficulty in nursing. On physical examination there was tympany on percussion of the left side of the chest and absent breath sounds; at times, gurgling sounds were heard in the chest. This child presented the signs and symptoms of diaphragmatic hernia usual in the new-born. The diagnosis was substantiated by roentgen examination and necropsy, which showed a left-sided, false hernia. In Fernandez's [39] case, the fourth diagnosed clinically, cyanosis, dyspnea and difficulty in breathing were the outstanding symptoms in a new-born infant. Physical examination showed dulness on percussion, absent breath sounds, bulging of the thorax and retraction of the abdomen. The diagnosis was confirmed by roentgenograms. This child also lived three months. Jansen [40] diagnosed a case clinically in a boy, aged 6 days; the outstanding symptoms were dyspnea and cyanosis. This was a false hernia on the right side, situated centrally. The diagnosis was substantiated by roentgen studies and necropsy.

38. Clark: Am. J. Obst. & Gynec. **8**:216 (Aug.) 1924.
39. Fernandez: Semana méd. **2**:1083 (Oct.) 1925.
40. Jansen: Nederl. Tijdschr. v. Geneesk. **2**:1796 (Oct.) 1927.

The sixth case diagnosed clinically was that of de la Vega,[41] in a boy, aged 7 years, whose chief complaints were attacks of dyspnea, cyanosis, diarrhea and vomiting, which always occurred at night. Physical examination revealed tympany on percussion of the left side of the chest; diminished breathing and gurgling sounds were heard in this side of the chest. The heart was displaced to the right. The patient had been seen by several other physicians, and a diagnosis of pulmonary tuberculosis with pleural effusion had been made. Thoracentesis had been performed twelve times. De la Vega saw the patient later and made a diagnosis of hernia based on the foregoing signs and symptoms. The diagnosis was substantiated by roentgenologic examinations.

Of the six cases diagnosed clinically, five were in infants, three, in new-born infants, all presenting characteristic signs and symptoms. The sixth case in a boy, aged 7 years, also presented signs and symptoms characteristic of hernia. A combination of circulatory and respiratory disturbances with difficulty in swallowing and vomiting should lead one to suspect diaphragmatic hernia in the new-born. This symptom complex is only produced, as a rule, by intrathoracic tumors, which are rare in children. In addition, changes in the percussion note and in the breath sounds, tinkling sounds and dextrocardia offer good confirmatory evidence. It is important not only to recognize the presence of hernia, but to make an effort as well to recognize the type, location and degree of herniation present: first, because of the prognosis and second, because of the possibility of surgical correction. For example, herniation of a small portion of the stomach through the esophageal opening is not incompatible with life and may present few symptoms (Friedenwald and Feldman's[42] six cases). In addition, as already stated, esophageal hernias lend themselves to surgical treatment more readily than any other type of hernia.

A study of the series of case reports collected reveals the fact that diaphragmatic hernia is more often confused with pleural effusion than with any other condition. This is especially true in older children, in whom diaphragmatic hernia must also be differentiated from subphrenic abscess with the accumulation of gas, and eventration of the diaphragm. In seven cases, diagnoses of hydropneumothorax, pyopneumothorax or just simple effusion were made; taps were performed in all seven cases. In two cases, more than one tap was performed, twelve being performed in de la Vega's case[41] and three in Brush's[43] case. The introduction of an aspirating needle into the chest cavity in the presence of a diaphragmatic hernia is not a harmless procedure and may be followed by death; for example, sudden death occurred in Reiss's[44] case after

41. De la Vega: Semana méd. **1**:548 (March) 1928.
42. Friedenwald and Feldman: Am. J. M. Sc. **170**:263 (Aug.) 1925.
43. Brush: Arch. Pediat. **44**:612, 1927.
44. Reiss: Jahrb. f. Kinderh. **77**:550, 1913.

the introduction of an aspirating needle; necropsy revealed the presence of the stomach and spleen in the left thoracic cavity. Hume [14] reported a case in which unusual chest signs were present on the left side; roentgenograms were made which were interpreted as showing the presence of a complete pneumothorax on the left side. Paracentesis was performed in the fifth left interspace; air bubbled out, and a small amount of brownish material was withdrawn. Four hours later, the patient suddenly died. Necropsy revealed a false hernia of the diaphragm with herniation of the stomach and ascending colon into the thoracic cavity. Even when death does not occur immediately, there is danger of the occurrence of pyopneumothorax and sepsis due to puncture of the herniated viscus.

Although in no cases in this series was a diagnosis of subphrenic abscess made, in older children there is a possibility of this diagnosis being made when hernia is present. The absence of constitutional symptoms and of pathologic processes, which may give rise to subphrenic abscess or pleural effusion, should lead one to rule out hernia of the diaphragm before resorting to exploratory puncture.

In regard to eventration of the diaphragm, it is impossible to make a differential diagnosis clinically between this condition and hernia, because the signs and symptoms in both conditions may be exactly alike. It is of extreme importance to make a differential diagnosis from the standpoint of surgical treatment, and resort to roentgenologic examination must be had. Indeed, Greenwald and Kaufman [45] stated that even roentgenologic examination is not always a deciding factor. This is borne out by the fact that in two cases in the series studied, a roentgenologic diagnosis of eventration was made when hernia existed (Schoenfeld,[8] Kienbock [6]). Careful studies in the lateral positions will reveal double contours of the diaphragm in eventration; this is not observed in hernia. Beltz [46] employed the introduction of air into the stomach by means of a catheter or stomach tube and observed the contour of the stomach fluoroscopically; in hernia the stomach, when distended, approaches the thoracic wall. The air is then aspirated while the patient is still behind the screen, and it is observed that the stomach moves away from the thoracic wall. The stomach also assumes various contours with the introduction or aspiration of air and with changes in the position of the patient. In eventration, however, a regular arch immediately over the stomach "air bubble" is always observed.

In two cases in this series an incorrect diagnosis of intussusception was made, because of vomiting and the presence of a mass in the epigastrium. The absence of discharges resembling raspberry jelly or a

45. Greenwald, H. M., and Kaufman, J.: Congenital Nonrotation of Stomach and Eventration, Am. J. Dis. Child. **35**:641 (April) 1928.

46. Beltz: München. med. Wchnschr., 1910, p. 1006.

thin, blood-stained, watery discharge containing no fecal matter should be sufficient reason to search for other lesions to account for the vomiting and the mass.

In the new-born, diaphragmatic hernia may be confused with intracranial hemorrhage, pyloric stenosis, congenital atelectasis of the lung, atresia of the esophagus and dextrocardia. When cyanosis, a weak feeble cry, respiratory difficulty and inability to nurse are present, it is possible to mistake hernia for intracranial hemorrhage. The presence of unusual signs in the chest and dextrocardia, however, in the absence of a bulging fontanel or convulsive seizures, should lead one to suspect hernia and to employ roentgenography to establish the diagnosis.

Because of the regurgitation frequently encountered in hernia, it may be mistaken for pyloric stenosis. The onset of symptoms before the infant is 2 or 3 weeks of age, the absence of visible peristalsis, the presence of a tympanitic or flat note on percussion of the lower part of the thorax coupled with respiratory difficulty should be sufficient to rule out stenosis. When any further doubt exists, a barium suspension should be injected into the stomach by means of a catheter, and a roentgenogram taken.

Atresia of the esophagus is practically always accompanied by a tracheal fistula. As a result, feeding is attended by violent coughing spells. A catheter passed into the esophagus meets with obstruction at some point above the cardia. Roentgenologic studies after the administration of opaque mediums may at times be necessary to differentiate the two conditions.

Four cases in this series were diagnosed as dextrocardia. Grulee [28] stated that the presence of dextrocardia in a new-born infant should always lead one to suspect diaphragmatic hernia, particularly when unusual chest signs are present. As a rule, true dextrocardia is accompanied by a complete situs inversus.

SUBSEQUENT COURSE AND PROGNOSIS

Diaphragmatic hernia may pursue one of three courses: (1) The infant may be stillborn or may die immediately after birth; (2) it may live a short time and then die of asphyxia or bronchopneumonia, or (3) the patient may grow up with varying symptoms and finally die suddenly because of strangulation of the herniated organ. It is also possible that the hernia may be latent, manifesting no symptoms during life. Under such conditions the hernia is discovered, as a rule, either by routine roentgenologic examination for some unrelated condition, or at necropsy, when death was due to some other cause.

The prognosis depends chiefly on the age of the patient. The incapacity of the infant suffering from this condition will depend, as a rule, on the size and extent of the hernia. If the hernial opening and contents

are small, the outlook is far better than when a large amount of intestine completely fills the thoracic cavity. In practically all cases in which death occurred immediately or soon after birth, the stomach, small intestine, a large portion of the colon and even the spleen and pancreas were herniated into the thoracic cacity. That death may occur even when one organ or a small portion of an organ is herniated can be seen from our case, in which the constant vomiting resulted in an aspiration pneumonia. As a general rule, however, it may be said that if the infant does not die soon after birth, the hernia will be well tolerated and the child may reach adult life, provided the herniated organ does not become strangulated. Indeed, Latta [7] cited cases in which congenital deficiencies of the diaphragm were present in patients who lived for from three days to seventy years. Fischer's patient lived three days; Millard's patient lived four days; Krombholz's, eleven days; Booker's, two months; Gilford's, four months; Hillier's, six months; Fothergill's, ten months; Lacher's, eleven and a half months; three lived three years (Textor, Mayes, two cases); three lived seven years (Diemenbröck, Textor, Downes); one lived eighteen years (Doberauer); one lived twenty-two years (Rosenthal, Keim, Huguier); one was a young adult (Beck); one lived thirty-one years (Hess); one lived thirty-three years (Abel); one lived forty-three years (Monks); one lived forty-seven years (Vogel); one lived fifty-seven years (Fry); one lived fifty-eight years (Carson and Huelsmann), and one lived seventy years (Guttman). All these authors were quoted by Latta.[7]

In new-born infants, the prognosis is decidedly bad. Of the eighty-two cases reviewed, forty-three were in new-born infants. Of these, thirteen were stillborn, thirteen lived less than one hour, nine lived less than twenty-four hours and two lived less than thirty-six hours. Death in these cases is due chiefly to interference with cardiac or pulmonary function, as is shown by the attending extreme cyanosis and respiratory difficulty.

In older children, the outlook is more encouraging. Of eighteen children over 1 year of age with hernia, four were operated on and recovered, and four were still living at the time the cases were reported. Five children died of intestinal obstruction, two died following thoracentesis, one died of cardiorespiratory failure, in one the cause of death was not stated and in one the subsequent course was not given. It will be noticed that in this age group, strangulation is the most common cause of death, in contrast to the younger age group in which cardiorespiratory failure is the cause of death.

TREATMENT

The treatment for this condition is obviously surgical. For the patient in whom the hernia makes its appearance at birth, little or nothing

can be done from a surgical standpoint. However, it may be possible in these cases to place the infant in a position that will be attended with a minimum amount of discomfort and vomiting. In our case, holding the infant in an upright position relieved the vomiting and cyanosis; this was probably due to a relaxation of the hernial ring about the herniated viscus. Davis [20] constructed a frame on which the infant was kept at an angle of 45 degrees and which gave the infant much relief and allowed nutrition and development to proceed normally. At 4 months of age, the infant was removed from the frame; subsequent to its removal, the child developed signs of intestinal obstruction and strangulation and died. Operation had been refused at 4 months of age. Thus, if the

TABLE 7.—*Patients Operated on with Recovery*

Year	Author	Age	Type	Side	Location	Result of Operation
1918	Downes	2 mos.	False	Right	Esophageal	Impossible to reduce stomach; gastro-enterostomy performed; patient symptom-free five months postoperatively
1918	Downes	7 yrs.	False	?	Esophageal	Anterior gastro-enterostomy; discharged symptomatically cured
1920	Griewe	5 yrs.	True	Left	Esophageal	Cured
1927	Jowers	7 mos.	?	Left	Posterior	Cured
1928	Pauchet	3 yrs.	True	Left	Esophageal	Cured

TABLE 8.—*Patients Operated on with Fatal Termination*

Year	Author	Age	Type	Side	Location	Result of Operation
1918	McCleave	3 mos.	False	Left	Posterior	Death a few hours postoperatively
1919	DeBuys	4 mos.	False	Left	Posterior	Death at operation
1921	Barnett	8 mos.	True	Left	?	Death ?
1927	Clark	3 yrs.	False	Left	Anterior	Death two hours postoperatively
1927	Leopold	4 yrs.	True	Left	Esophageal	Death four hours postoperatively
1927	Olmstead	6 mos.	False	Left	Central	Death nine hours postoperatively

vomiting can be controlled by careful and constant attention, it may be possible to carry the infant along until surgical treatment is feasible.

In infants several months old, if symptoms are present, it is advisable not to defer surgical treatment because of the danger of increase in the amount of herniated viscus as time goes on. In McCleave's [18] patient, operation was delayed, thus allowing more of the intestine to become herniated and thereby occasioning much more manipulation at operation and consequent shock. The defect itself was readily amenable to operation.

A study of the cases in our series in which operation was performed shows the following: eleven patients were operated on, with five recoveries (45.4 per cent) and six deaths (54.4 per cent). Of the five operations that were successful, four were esophageal and one posterior in location (tables 7 and 8). It seems justifiable, therefore, to conclude

Résumé of Cases Collected from the Literature (1912-1928, Inclusive)

No.	Year	Author	Sex	Age	Side	Type	Size	Position	Comment
1	1912	Berenguer: Semana méd. **19**: 549	M	New-born	Left	?	3 cm.	Posterior	Diagnosis made at necropsy; clinical diagnosis: ?; symptoms: attacks of dyspnea and cyanosis a few hours after birth; signs: dextrocardia; course: death 32 hours after delivery; contents of hernia: small intestine, part of large intestine, stomach
2	1912	Devrraigne: Bull. Soc. d'obst.et de gynéc. de Paris **1**: 458	?	New-born	Left	False	Huge	Posterior	Diagnosis made at necropsy; clinical diagnosis: intracranial hemorrhage; symptoms: failure to breathe, heart beat present for one hour after delivery; course: death one hour after delivery; contents: stomach, small intestine, part of colon
3	1913	Rischbieth: Australasian M. Gaz. **33**: 359	F	Stillborn	Left and right	False and true	?	Complete absence of left leaflet, esophageal, right	Diagnosis made at autopsy; clinical diagnosis: ?; symptoms: stillbirth; contents: left side—stomach, spleen, pancreas, great omentum and small intestine; right side—lobe of liver, head of pancreas
4	1913	Poteezki [58]	?	New-born	Right	False	Large	Posterior	?
5	1913	Bulloch: Indian M. Gaz. **48**: 271	M	New-born	Left	False	?	Complete absence of left leaflet	Diagnosis made at autopsy; clinical diagnosis: ?; symptoms: feeble attempts at respiration at intervals, cyanosis; signs, ?; course: death one hour after delivery; contents: left lobe of liver, stomach, small intestine, part of large intestine, spleen
6	1913	Reiss [44]	?	3 years	Left	True	Large	Esophageal opening	Diagnosis made at autopsy; clinical diagnosis: pneumonia and pyopneumothorax; roentgen diagnosis: pneumonia and pyopneumothorax; symptoms: cough treated two years for "bronchitis"; course: thoracentesis, sudden death; contents: stomach, spleen
7	1914	Fletcher: Australasian M. Gaz. **48**: 271, 1913	?	2 years	Left	?	2 fingers in circumference	Central	Diagnosis made at necropsy; clinical diagnosis: ?; symptoms: none seven hours before death, acute onset—dyspnea, cyanosis, restlessness; course: death due to obstruction, 7 hours; contents: stomach, omentum
8	1914	Oui and Devuldeen: Bull. Soc. d'obst. et de gynéc. de Paris **111**: 545	?	New-born	Both sides	False	Large	?	Diagnosis made at necropsy; clinical diagnosis, ?; symptoms: breathed once, became cyanotic, death; contents: liver
9	1914	Fidler [35]	?	8 months	Left	True	?	Posterior and central	Diagnosis made at necropsy; clinical diagnosis: ?; roentgen diagnosis circumscribed air pocket filling entire left side of thorax; symptoms: grayish cyanosis; signs: left side of chest bulging, tympanitic note on left side from second rib downward, bronchial breathing; course: at 2 years of age, evidence of pneumonia followed by death; contents: stomach, aplasia of left lung, pneumonia of right lung
10	1915	Grulee [28]	?	New-born	Left	False	Small	Central	Diagnosis: intravitam by x-ray; clinical diagnosis: dextrocardia; symptoms: ?; course: death; diagnosis proved by autopsy
11	1915	Monti: Wien. klin. Wchnschr. **28**: 788	?	4 months	Left	?	?	?	Diagnosis: intravitam by x-ray; clinical diagnosis: ?; symptoms: cough, dyspnea, cyanotic spells during feeding, vomiting spells six weeks' duration, failure to gain in weight; signs: dextrocardia, tympany on percussion, diminished breath sounds; contents: ?

12	1915	Kienböck [6]	?	13 months	Left	False	?	?	Diagnosis: intravitam by x-ray; clinical diagnosis: ?; roentgen diagnosis: eventration—subsequent diagnosis made by x-ray; symptoms: pallor, weakness since birth; at 8 months, dyspnea, cough, difficulty in swallowing, vomiting after food; noise heard in chest when food is taken; signs: symmetrical bulging of thorax, dyspnea, dulness on percussion of left side posteriorly, dextrocardia; contents: stomach and intestines
13	1916	Rüder [25]	?	New-born	Left	?	3 cm.	?	Diagnosis made at necropsy; clinical diagnosis, ?; symptoms: cried lustily at birth; collapse and death after severing cord; signs: ?; contents: stomach and intestine
14	1917	McCleave [18]	M	3 months	Left	False	2 cm.	Posterior	Diagnosis: intravitam by x-ray; clinical diagnosis: ?; roentgen diagnosis: diaphragmatic hernia; symptoms: cyanosis, dyspnea, pain relieved by vomiting; onset at two weeks; signs: tympanitic note on percussion of left side of chest, retraction of abdomen, dextrocardia; course: operation followed by death after a few hours; contents: intestines
15	1917	Cockayne: J. Anat. **52**: 64	?	Fetus	Left	False	2½ cm.	Posterior	Diagnosis made at autopsy; stillbirth; contents: two accessory lungs and accessory spleen
16	1918	Bund: Frankfurt Ztschr. f. Path. **21**: 243	?	8 months	Right	True	?	Esophageal	Diagnosis: ?; clinical diagnosis: ?; symptoms: ?; contents: stomach in right posterior mediastinum
17	1918	Gross: J. M. Research **38**: 473 (July)	M	Stillborn	Left	True	Complete absence of left leaflet	?	Diagnosis at necropsy: stillbirth; contents: anomalous lobe of liver, stomach, omentum, small intestine, spleen and pancreas
18	1918	Fisk, quoted by Richards: Ann. Otol Rhin. & Laryng. **32**: 1145	M	New-born	Left	False	Large	Posterior	Diagnosis: ?
19	1918	Schafer [33]	F	3 months	Right	False	3 f.	Anterior	Diagnosis made at necropsy; clinical diagnosis, ?; roentgen diagnosis: plates could not be interpreted; symptoms: crying, vomiting, frequent stools, cough, dyspnea; signs: diminished expansion on right side of chest, marked tympany on right side of chest, râles; course: ?; contents: stomach, intestine, part of pancreas, part of large intestine
20	1918	Downes: Surg. Gynec. Obst. **27**: 393 (Oct.)	M	7 years	?	False	3 cm.	Esophageal	Diagnosis: intravitam by x-ray; clinical diagnosis: ?; roentgen diagnosis: hernia; symptoms: five years' duration, periodic vomiting, failure to gain, discomfort after meals; signs: ?; course: operation, impossible to reduce stomach, gastro-enterostomy performed, leaving stomach in thoracic cavity—patient discharged symptom-free; contents: stomach and duodenum
21	1918	Downes: Surg. Gynec. Obst. **27**: 393 (Oct.)	?	20 months	Right	False	2 inches	Esophageal	Diagnosis made at operation; clinical diagnosis: ?; symptoms: ?; course: operation, impossible to reduce stomach because of adhesions; gastro-enterostomy performed; 5 months postoperative, no symptoms, gain of 10 pounds
22	1919	MacCandless: J. Roentgenol. **2**: 282 (March)	?	7 weeks	Right	?	?	?	Diagnosis: intravitam by x-ray; clinical diagnosis: ?; roentgen diagnosis: hernia; symptoms: paroxysmal dyspnea, constipation, cold sweats; signs: displacement of heart to left, tympany on percussion of right side of chest, epigastric bulge; course: living at end of 12 weeks; contents: small intestine

Résumé of Cases Collected from the Literature (1912-1928, Inclusive)—Continued

No.	Year	Author	Sex	Age	Side	Type	Size	Position	Comment
23	1919	DeBuys [24]	F	4 months	Left	False	?	Posterior	Diagnosis made at operation; clinical diagnosis: intussusception; symptoms: attacks of colic relieved by enema, vomiting; signs: immobility of left side of chest, tympany on percussion of left side of chest, absent breath and voice sounds in left side of chest, scaphoid abdomen; course: operation for intussusception—death shortly after operation; diagnosis confirmed by autopsy; contents: stomach, colon, large omentum
24	1920	Carmichael, quoted by Richards: Ann. Otol. Rhin. & Laryng. **32**: 1145	?	New-born	Left	?	?	Posterior	Diagnosis: ?
25	1920	Mayes: M. Rec. **97**: 713	?	New-born	Left	False	2 by 5 cm.	Posterior	Diagnosis made at autopsy; clinical diagnosis, ?; course: death from asphyxiation; contents: left kidney
26	1920	Griewe [26]	F	5 years	Left	True	5 by 6 cm.	Esophageal	Diagnosis: intravitam by x-ray; clinical diagnosis: pyloric obstruction; roentgen diagnosis: hernia; symptoms: vomiting since birth, constipation, failure to gain in weight; signs: ?; course: operation, recovery; contents: stomach
27	1921	Fernandez: J. Philippine Islands M. A. **1**: 12	M	8 months	Left	?	?	?	Diagnosis made at necropsy; clinical diagnosis: ?; symptoms: vomiting, occasional cough; signs: ?; contents: small intestine, large intestine, pancreas
28	1921	Jalin: Ztschr. f. d. ges. Anat. **61**: 165	F	Fetus	Left	False	3 by 5 cm.	?	Diagnosis made at autopsy: stillbirth; contents: ?
29	1921	Cohn and Czempin: Zentralbl. f. Gynäk. **45**: 1490 (Oct.)	F	New-born	Right	False	2 fingers	Posterior	Diagnosis made at autopsy; clinical diagnosis, ?; symptoms: convulsive attempts at respiration; signs: ?; course: death 2 hours after delivery; contents: jejunum and portion of colon
30	1921	Barnett: Brit. M. J. **1**: 602 (April)	?	8 months	Left	True	Large	?	Diagnosis made at operation, confirmed at autopsy; clinical diagnosis: intestinal obstruction; symptoms: vomiting, constipation, distention of abdomen, cyanosis; signs: ?; course: operation, death
31	1921	Morel: Lyon chir. **18**: 730	M	Stillborn	Left	False	Large	?	Diagnosis made at autopsy: stillbirth; contents: stomach, small intestine
32	1921	Hofer: Arch. f. Gynäk. **114**: 620	?	New-born	Left	False	Large	?	Diagnosis made at necropsy; clinical diagnosis: ?; symptoms: failure to breath at birth, strong heart beat present at birth, death after 1½ hours; signs: ?; contents: left lobe of liver, stomach, jejunum
33	1921	Hofer	?	Stillborn	Right	False	Large	?	Diagnosis made at necropsy: stillbirth; contents: liver
34	1921	Cleiz: Bull. et mém. Soc. anat. de Paris **91**: 101	F	New-born	Left	False	2 fingers	Posterior	Diagnosis made at autopsy; clinical diagnosis: ?; symptoms: made several attempts at respiration, single weak cry; signs: ?; course: death 1¾ hours after delivery; contents: stomach, small intestine, part of colon
35	1922	VonDongen [28]	?	New-born	Left	False	4 cm.	Posterior	Diagnosis made at autopsy; clinical diagnosis: dextrocardia; symptoms: peculiar cry immediately after delivery; made little effort at respiration; cyanosis; heart beat present for ½ hour; signs: dextrocardia; course: death in ½ hour; contents: stomach, duodenum, part of colon, spleen

36	1922	Schreiber [22]	?	New-born	Left	False	Absence of left leaflet	?	Diagnosis: intravitam clinically, confirmed by x-ray and necropsy; clinical diagnosis: hernia, based on unexplained cyanosis, peculiar cry and dextrocardia; roentgen diagnosis: hernia; symptoms: peculiar cry—one cry, then became very cyanotic and ceased to breathe; heart beat persisted and was to right; signs: dextrocardia; course: death directly after delivery; contents: stomach, small intestine, spleen, left lobe of liver
37	1922	Hume [14]	M	6 months	Left	False	2½ inches	Posterior	Diagnosis made at autopsy; clinical diagnosis: complete left pneumothorax; roentgen diagnosis: complete left pneumothorax; symptoms: acute onset—abdominal pain, vomiting, temperature 97 F., pulse 104, respirations 40; signs: succussion splash, metallic sound and "bell" sounds heard in left side of chest; course: thoracentesis thoracis followed by death; contents: stomach, ascending colon, omentum
38	1922	Hume [14]	?	Fetus	Left	False	Complete absence of left leaflet	?	Diagnosis made at autopsy: stillbirth; contents: stomach, small intestine, part of colon
39	1922	Blumenfeld: New York M. J. **116**: 131 (Aug.)	?	New-born	Left	?	Slit in diaphragm	Posterior	Diagnosis made at autopsy; clinical diagnosis: ?; symptoms: faint cry and gasp at birth, labored attempts at respiration, cyanosis; signs: ?; course: death 1½ hours after delivery; contents: stomach, large and small intestines
40	1922	Davis [20]	M	8 days	Left	?	?	?	Diagnosis: intravitam clinically, confirmed by x-ray; clinical diagnosis: hernia; roentgen diagnosis: hernia; symptoms: transitory attacks of cyanosis and dyspnea, relieved by holding child in upright position, cyanosis intense after feeding; signs: bulging of thorax, retraction of abdomen, tympanitic note on percussion of left side of chest, absent breath sounds in left side of chest; course: patient did well until fourth month—kept on frame at angle of 45 degrees, operation refused at fourth month—child taken off frame, suddenly developed strangulation of intestine, resulting in death; contents: small and large intestines
41	1922	Latta [7]	M	New-born	Left	False	Complete absence of left leaflet	?	Diagnosis made at autopsy; clinical diagnosis: ?; symptoms: cyanosis, unsuccessful attempts at respiration; signs: ?; course: death shortly after delivery; contents: jejunum and stomach
42	1923	Adeney: Brit. M. J. **2**: 415 (Sept.)	?	New-born	Left	?	3 fingers	Posterior	Diagnosis made at autopsy; clinical diagnosis: ?; symptoms: ?; signs: ?; course: death 3 hours after delivery; contents: small and large intestine, spleen
43	1923	Stimson: Arch. Pediat. **40**: 647 (Oct.)	M	11 months	Right	?	?	Posterior	Diagnosis: intravitam by x-ray; clinical diagnosis: ?; roentgen diagnosis: hernia; symptoms: vomiting since third day of life, malnutrition, no cyanosis, no dyspnea; signs: dulness at right base posteriorly, diminished breath sounds on right side; course: living at age of 29 months; contents: stomach and duodenum
44	1924	Swope: Radiology **2**: 260 (April)	?	4 days	Left	?	?	?	Diagnosis: intravitam by x-ray, confirmed by autopsy; clinical diagnosis: ?; roentgen diagnosis: hernia; symptoms: ?; signs: ?; contents: jejunum and part of large intestine

Résumé of Cases Collected from the Literature (1912-1928, Inclusive)—Continued

No.	Year	Author	Sex	Age	Side	Type	Size	Position	Comment
45	1924	Barney and Evans: Arch. Pediat. **41**: 720 (Oct.)	?	New-born	Right	False	Large	?	Diagnosis made at necropsy; clinical diagnosis: ?; symptoms: marked cyanosis, unsuccessful attempts at respiration; signs: ?; course: death soon after delivery; contents: small intestine, part of colon
46	1924	Barney and Evans.....	?	New-born	Right	False	Large	?	Diagnosis made at autopsy; clinical diagnosis: ?; symptoms: ?; signs: heart displaced to left; course: death soon after delivery; contents: liver
47	1924	Southby [32]	M	2½ years	Right	True	?	Posterior	Diagnosis made at necropsy; clinical diagnosis: severe anemia of unknown origin; roentgen diagnosis: tuberculous process in right lung; symptoms: pallor, weakness since birth; signs: gurgling sounds heard in abdomen, tympany on percussion of right side of chest, normal breath sounds; course: five months later readmitted with signs of bronchopneumonia plus marked cyanosis—death; contents: stomach and duodenum
48	1924	Tibbets: Brit. M. J. **1**: 236 (Feb.)	?	New-born	Right	False	3 f.	?	Diagnosis made at autopsy: clinical diagnosis: ?; symptoms: feeble cry, cyanosis; signs: ?; course: death 1 hour after delivery; contents: stomach, duodenum
49	1924	Clark [38]	?	New-born	Left	False	3 cm.	?	Diagnosis made at necropsy; clinical diagnosis: ?; symptoms: recurrent attacks of cyanosis and dyspnea, nursed well except during an attack of dyspnea; signs: ?; course: died suddenly at end of two weeks; contents: stomach, small intestine, ascending and transverse colon
50	1924	Clark [38]	?	New-born	Left	False	?	Posterior	Diagnosis: intravitam, clinically confirmed by x-ray and necropsy; clinical diagnosis: hernia; roentgen diagnosis: hernia; symptoms: cyanosis, weak moaning cry, dyspnea, nursed with difficulty; signs: left side of chest: tympanitic note, signs of cavitation at times, absent breath sounds over entire left side, at times gurgling sounds heard in left side of chest; course: death at age of 6 days; contents: jejunum, ileum, ascending and transverse colon
51	1924	McCabe: Brit. M. J. **2**: 256 (Aug.)	F	2 years	Left	?	½ inch in diameter	Posterior	Diagnosis made at autopsy; clinical diagnosis: ?; symptoms: vomiting—onset sudden; signs: marked epigastric pulsation; course: death two days after onset; contents: stomach
52	1924	Hess: M. Clin. N. Amer. **8**: 567 (Sept.)	?	New-born	Left	False	?	Posterior	Diagnosis made at autopsy; clinical diagnosis: ?; symptoms: ?; course: death six hours after delivery; contents: jejunum, ileum, part of colon
53	1924	Hess	M	6½ years	Left	True	?	?	Diagnosis: intravitam by x-ray; clinical diagnosis: ?; roentgen diagnosis: hernia; symptoms: present since infancy, restlessness, sharp cry as if in pain, underweight; signs: failure of left side of chest to move with respiration, underdevelopment of left side, flatness at base, tympany above, dulness at apex, absent breath sounds, dextrocardia; course: still living; contents: stomach, small intestine and colon
54	1924	Hess	M	3 years	Left	True	?	?	Diagnosis: intravitam by x-ray; clinical diagnosis: ?; symptoms: accidentally found during course of pneumonia on right side at 8½ months, cyanosis, underdevelopment; signs: tympany on left side, absent breath sounds, diminished expansion and underdevelopment of left side of chest, dextrocardia; course: still living at end of five years; contents: stomach, splenic flexure of colon

55	1924	Hess	M	8 weeks	Left	False	4 cm.	Posterior	Diagnosis made at necropsy; clinical diagnosis: situs invertus, congenital atelectasis of left lung; symptoms: repeated cyanotic spells, short attacks of coma, at times associated with vomiting, constipation; signs: rapid heart beat, absent breath sounds, tympanitic note over left side of chest, dextrocardia; course: at age of 14 weeks baby given bottle, followed by a severe cyanotic spell, coma and death; contents: small intestine, part of large colon
56	1925	Fernandez [39]	M	New-born	Right	False	4 cm.	Central	Diagnosis made at autopsy; clinical diagnosis: ?; symptoms: cyanosis, ineffective attempts at respiration; signs: ?; course: death one half hour after delivery; contents: part of right lobe of liver, small intestine
57	1925	Fernandez [39]	?	New-born	Left	?	?	?	Diagnosis: intravitam, confirmed by x-ray; clinical diagnosis: hernia; roentgen diagnosis: hernia; symptoms: cyanosis, increased at feeding, difficulty in feeding, dyspnea; signs: absent breath sounds, dulness in left side of chest, bulging of left side of thorax; course: living at end of three months; contents: stomach
58	1926	Cruikshank: Glasgow M. J. **105**: 81 (Feb.)	F	Stillborn	Left	?	?	Posterior	Diagnosis made at autopsy: stillbirth; contents: left lobe of liver, pancreas, stomach, small intestine, cecum, part of colon
59	1926	Cruikshank	F	Stillborn	Left	?	?	Posterior	Diagnosis made at autopsy: stillbirth; contents: stomach, small intestine, spleen
60	1926	Dreyer: Illinois M. J. **50**: 47 (July)	?	Stillborn	Left	?	Large	Posterior	Diagnosis made at autopsy: stillbirth; contents: "viscera in pleural cavity"
61	1926	Unger and Speiser: Am. J. Roentgenol. **15**: 125 (Feb.)	M	New-born	Left	?	?	Central	Diagnosis: intravitam by x-ray, confirmed by autopsy; clinical diagnosis: ?; roentgen diagnosis: hernia; symptoms: failure to breathe, cyanosis, weak feeble cry; signs: ?; course: death seven hours after delivery; contents: stomach, spleen, small intestine and cecum
62	1926	Unger and Speiser.....	M	New-born	Left	?	?	Central	Diagnosis: intravitam by x-ray, confirmed by autopsy; clinical diagnosis: ?; roentgen diagnosis: hernia; symptoms: failure to breathe, weak feeble cry, cyanosis; signs: ?; course: death seven hours after delivery; contents: stomach, spleen, small intestine and cecum
63	1926	Unger and Speiser.....	M	1½ years	Left	?	1 inch	?	Diagnosis made at necropsy; clinical diagnosis: congenital syphilis; symptoms: pallor; signs: impaired resonance, diminished breath sounds over left lower side of chest, liver and spleen enlarged; course: ?; contents: small and large intestines
64	1926	Unger and Speiser.....	M	1 year	Left	?	4 fingers	Posterior	Diagnosis: intravitam by x-ray; clinical diagnosis: pleural effusion; roentgen diagnosis: hernia; symptoms: dyspnea, cyanosis, vomiting, diarrhea, inability to take food; course: thoracentesis, death; contents: intestine, one half of left kidney
65	1926	Dondarini: Pediatria **33**: 1293 (Dec.)	?	New-born	Left	False	Large	?	Diagnosis made at autopsy; clinical diagnosis: ?; symptoms: fifteen hours after delivery, dyspnea, cyanosis, especially when crying; signs: dextrocardia, tympanitic note in left side of chest; course: sudden death soon after onset of symptoms; contents: stomach, small intestine, part of large intestine
66	1926	Schönfeld [8]	F	7 days	Left	True	4 by 5 cm.	?	Diagnosis made at autopsy; clinical diagnosis: dextrocardia; roentgen diagnosis: eventration; symptoms: cyanosis when crying; signs: dextrocardia, absent breath sounds on left side of chest; contents: left lobe of liver, stomach

Résumé of Cases Collected from the Literature (1912-1928, Inclusive)—Continued

No.	Year	Author	Sex	Age	Side	Type	Size	Position	Comment
67	1926	Ohnell: Acta radiol. **6**: 3	M	3¾ years	Left	?	?	Esophageal	Diagnosis: intravitam by x-ray; clinical diagnosis: ?; roentgen diagnosis: hernia; symptoms: frequent, profuse vomiting, immediately after meals since birth, failure to gain weight; signs: ?; contents: ?
68	1926	McEachern: Arch. Pediat. **43**: 688 (Oct.)	?	18 days	?	True	?	Esophageal	Diagnosis made at autopsy; clinical diagnosis: pyloric stenosis; symptoms: cough, vomiting, loss of weight, cyanosis; signs: ?; contents: stomach
69	1927	Clark: Illinois M. J. **52**: 63 (July)	F	3½ years	Left	False	3½ by 2 cm.	Anterior	Diagnosis: intravitam by x-ray, confirmed by operation; clinical diagnosis: ?; symptoms: epigastric pain, vomiting; signs: retracted abdomen, dehydration, anemia; course: operation, death two hours postoperative; contents: omentum, stomach, small intestine, transverse colon, spleen
70	1927	Jung: Bull. Soc. d'obst. et de gynéc. de Paris **16**: 237 (March)	?	New-born	Left	?	?	?	Diagnosis made at autopsy: signs: death immediately after delivery; contents: ?
71	1927	Jansen [40]	M	6 days	Right	False	Large	Central	Diagnosis: intravitam clinically, confirmed by x-ray and autopsy; clinical diagnosis: hernnia; roentgen diagnosis: hernia; symptoms: dyspnea and cyanosis; signs: ?; contents: small intestine, part of large intestine
72	1927	Leopold: Am. J. Dis. Child. **33**: 597 (April)	M	4 years	Left	True	3 fingers	Esophageal	Diagnosis: intravitam by x-ray, confirmed by operation; clinical diagnosis: ?; symptoms: abdominal pain, discomfort after meals, constipation, loss of weight, later symptoms of intestinal obstruction; signs: ?; course: operation, death four hours postoperative; contents: stomach, small intestine, part of colon
73	1927	Tyson: Arch. Pediat. **44**: 557 (Sept.)	M	7 days	Left	?	1½ by 2 cm.	Posterior	Diagnosis: intravitam by x-ray, confirmed by autopsy; clinical diagnosis: dextrocardia, collapse of lung; symptoms: cyanosis, rapid irregular respirations; signs: dextrocardia; contents: small intestine, ascending and transverse colon, superior pole of left kidney
74	1927	Brush [43]	M	9 months	Left	?	?	?	Diagnosis: intravitam by x-ray; clinical diagnosis: bronchostenosis; roentgen diagnosis: first picture led to diagnosis of bronchostenosis, subsequent studies led to diagnosis of hernia; symptoms: vomiting, alternated diarrhea and constipation, chronic cough, convulsions, sudden attack of dyspnea and cyanosis, loss of consciousness; signs: bulging in left side of chest, flatness of tympanitic note alternating on left side of chest on percussion; breath sounds distant and bronchial in character; dextrocardia; course: three dry chest taps before diagnosis; has remained well at age of 2½ years; becomes cyanotic when excited; contents: ?
75	1927	De la Vega [41].....	M	7 years	Left	?	?	?	Diagnosis: intravitam clinically, confirmed by x-ray; clinical diagnosis: hernia—previous diagnosis, pulmonary tuberculosis; roentgen diagnosis: hernia—previous diagnosis, pulmonary tuberculosis; symptoms: attack of dyspnea and cyanosis, vomiting, always at night, diarrhea, temperature 102 F., complaints since 4 days of age; signs: tympany on percussion of left side of chest, diminished breath sounds on left side of chest; borborygmus dextrocardia; course: twelve pleural punctures before diagnosis was made—still living at time case was reported; contents: small intestine and part of large intestine

76	Jowers:	Brit. J. Surg. **15**: 332 (Oct.)	?	7 months	Left	?	?	Posterior	Diagnosis at operation; clinical diagnosis: intussusception; symptoms: pain in epigastrium, vomiting, later severe paroxysms of pain; signs: mass in epigastrium to left of midline; course: operation revealed true diagnosis—repair and recovery; contents: stomach
77	1927	Waldron: Brit. M. J. **2**: 1225 (Dec.)	?	New-born	Left	?	3 inches	Posterior	Diagnosis made at autopsy; clinical diagnosis: ?; symptoms: face blue black immediately after birth, respirations short and jerky, four per minute; signs: abdomen distended and tense, dextrocardia; course: death 15 minutes after delivery; contents: small intestine, spleen, part of colon
78	1927	Argonz and Ruiz: Semana méd. **1**: 109 (Jan.)	?	New-born	Left	?	?	?	Diagnosis made at autopsy; clinical diagnosis: ?; symptoms: cyanosis, difficulty in breathing; signs: ?; course: death 20 hours after delivery
79	1927	Guistinian [37]	?	22 months	Right	?	?	Posterior	Diagnosis made at autopsy; clinical diagnosis: pleural effusions right side; roentgen diagnosis: small foci of hepatization; symptoms: underweight, dyspepsia, fever; signs: asymmetry of thorax, lack of development of right side of chest, diminished excursion of right side of chest; right side—diminished breath sounds, râles, dulness; course: thoracentesis—turbid fluid removed—death; contents: small intestine, cecum, appendix, ascending and transverse colon
80	1928	Pauchet and Luguet [31]..	?	3 years	Left	True	?	Esophageal	Diagnosis made at operation; clinical diagnosis: ?; roentgen diagnosis: esophageal diverticulum; symptoms: epigastric pain, vomiting since infancy, symptoms were intermittent, habitual constipation, poor appetite, hematemesis; signs: ?; course: operation, recovery; contents: ?
81	1928	Olmstead [36]	F	6 months	Left	?	?	Central	Diagnosis: intravitam by x-ray, confirmed by operation; clinical diagnosis: mediastinal tumor, pleural effusion; roentgen diagnosis: first diagnosis "lower one half chest homogeneous shadow, upper ½ multilocular-air containing shadow, pleural effusion—encapsulated," later diagnosis, hernia; symptoms: at age of 4 months, anorexia with constipation, at 5 months, cough difficulty in breathing, later, cyanosis, dyspnea, tinkling sounds after food; signs: dulness on percussion of left side of chest, anteriorly and posteriorly, diminished breath sounds, tubular breathing; course: after first roentgen diagnosis: thoracentesis, removal of fluid resembling chyle; later, operation, death nine hours postoperative
82	1928	Greenwald and Steiner...	M	2 days	Right	True	1 cent.	Esophageal	Diagnosis: intravitam by x-ray, confirmed by necropsy; clinical diagnosis: eventration of diaphragm; roentgen diagnosis: hernia; symptoms: dyspnea, cyanosis after a feeding, vomiting; signs: tympanitic note on percussion of left side of chest, diminished breath sounds; course: death from aspiration pneumonia six days after birth; contents: stomach

that hernias through the esophageal opening offer a better outlook in that they are more amenable to operation. Of the six patients who died, in only one was the hernia through the esophageal opening. Four of the five patients who recovered were children over 1 year of age; the youngest was 7 months of age. Of the six who died only two were over 1 year of age and four were under 1 year of age, showing that the older the child, the better the operative risk.

SUMMARY

1. Diaphragmatic hernia is not as infrequent as is generally believed.

2. A review of the literature of all cases in infants and children reported since 1912 is presented and the various cases analyzed.

3. Particular attention has been paid to symptomatology, diagnosis and differential diagnosis in an effort to facilitate recognition of the condition.

4. An effort has been made to divide diaphragmatic hernia into three groups, depending on the age of the patient and the predominating symptoms.

5. An additional case of true nontraumatic diaphragmatic hernia of the right side in a new-born infant is presented; the diagnosis was made during life and was confirmed at necropsy.

Pediatric Biographies

THOMAS SYDENHAM
1624-1689

JOHN RUHRÄH, M.D.
BALTIMORE

Sydenham is one of the best known names in the history of medicine. The biography of Sydenham has been written by Payne, by Latham, by George Wallis and by Fredéric Picard. The essay on "Locke and Sydenham" by John Brown in "Horae Subsecivae" is well worth reading, and there are innumerable items in which he is more briefly mentioned.

Sydenham came from a Puritan family, and was born in Dorsetshire at a small village called Wynford Eagle. His family dates back to King John; it furnished divines and jurists, but found its full flowering in Thomas. In 1642, at 18 years of age, he was matriculated as a Fellow Commoner in Magdalen Hall, Oxford, but his student career was cut short by the Civil War. In 1646, he returned to his university life, and it is related that on his way back to Oxford he fell in with Dr. Thomas Coxe who was attending his brother, and was persuaded to study medicine. This time Sydenham was enrolled in Wadham College. He received an M. A. degree and was "created" a Bachelor of Medicine by order of the Earl of Pembroke, on April 14, 1648. This sounds strange, but there are numerous instances in which physicians received their degrees by royal or other authority. An interesting example is that of John Shaw, who received an M. D. degree by act of the General Assembly of Maryland. Sydenham then received a fellowship in All Souls' College. There were no opportunities for clinical study, only for the study of the classics, chemistry and anatomy, and not much opportunity for the study of the last two.

After leaving Oxford, in 1655, he settled in London in Westminster, where he apparently mixed a bit in politics and was made "Comptroller of the Pike." Later he went to Montpelier to attend the clinical lectures of Charles Barbeirac. He returned to London in 1661, and was admitted to a licentiate of the College of Physicians in 1663, but he never obtained a fellowship. At the time of the plague he left London, and while he was away he wrote his first medical treatise, which was followed by some other writings. His works were written in Latin, evidently with the assistance of others better versed in that tongue. They were translated into English, the translation of John Pechey being the easiest to

read. Sydenham died in 1689 and was buried in St. James Church, Westminster. The inscription on his tombstone reads:

Prope hunc locum sepultus est
Thomas Sydenham.
medicus in omne aevum nobilis
notus erat A.D. 1624.
vixit annos 65.

A physician famous for all time, as the inscription says.

Sydenham has been called the English Hippocrates. One has only to compare his clinical histories, his descriptions of epidemics and of disease as it came under his observation with most of the preceding or contemporaneous medical writing to see how much more practical he was. He hated long-winded discussion of theories and devoted himself to the facts as he saw them. His views on the nature and variation of epidemics were sound, and he believed that certain diseases would disappear and others take their place, which has been amply demonstrated.

His pediatric contributions were important. He definitely separated chorea, often called Sydenham's chorea, from the chorea major or St. Vitus' dance of the Middle Ages. He described scarlet fever, but omitted the sore throat and did not recognize that the condition was contagious. He also described measles.

One paragraph on chorea is as follows:

This is a kind of convulsion, which attacks boys and girls from the tenth year to the time of puberty. It first shows itself by limping or unsteadiness in one of the legs, which the patient drags. The hand cannot be steady for a moment. It passes from one position to another by a convulsive movement, however much the patient may strive to the contrary. Before he can raise a cup to his lips, he makes as many gesticulations as a mountebank; since he does not move it in a straight line, but has his hand drawn aside by spasms, until by some good fortune he brings it at last to his mouth. He then gulps it off at once, so suddenly and so greedily as to look as if he were trying to amuse the lookers-on.

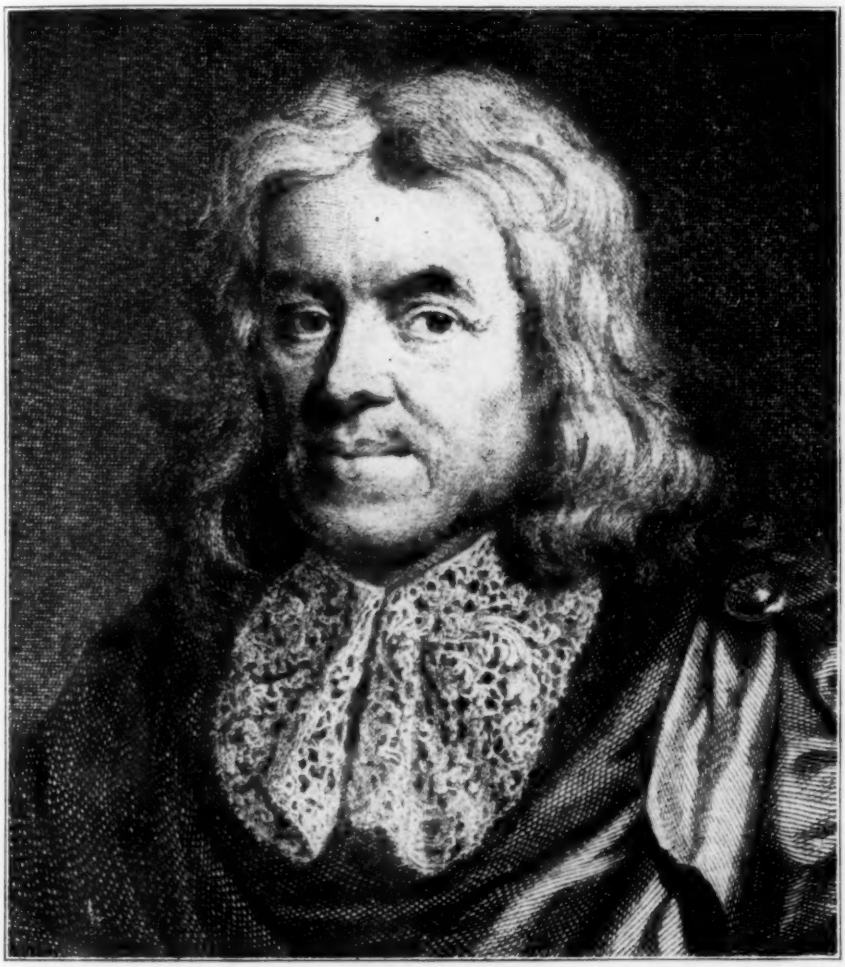
An extract from his account of scarlet fever must suffice:

Scarlet-fever (Scarlatina) may appear at any season. Nevertheless, it oftenest breaks out towards the end of summer, when it attacks whole families at once, and more especially the infant part of them. The patients feel rigors and shiverings, just as they do in other fevers. The symptoms, however, are moderate. Afterwards, however, the whole skin becomes covered with small red maculae, thicker than those of measles, as well as broader, redder, and less uniform. These last for two or three days, and then disappear. The cuticle peels off; and branny scales remain, lying upon the surface like meal. They appear and disappear two or three times.

A part of his description of measles is a bit of fine clinical writing:

This disease begins and ends within the above-named period. It generally attacks infants, and, with them, runs through the whole family. It begins with

shiverings and shakings, and with an inequality of heat and cold, which, during the first day, mutually succeed each other. By the second day, this has terminated in a genuine fever, accompanied with general disorder, thirst, want of appetite, white (but not dry) tongue, slight cough, heaviness of the head and eyes, and continued drowsiness. Generally there is a weeping from the eyes and nostrils; and this epiphora passes for one of the surest signs of the accession of the com-



Thomas Sydenham.

plaint. But to this may be added another sign equally sure; viz., the character of the eruption. Although measles usually shows itself by an exanthema upon the face, there appears upon the breast a second sort of breaking-out. This consists in broad red patches on a level with the skin, rather than true exanthemata. The patient sneezes as if from cold, his eyelids (a little before the eruption) become puffy; sometimes he vomits: oftener he has a looseness; the stools being greenish.

This last symptom is commonest with infants teething, who also are more cross than usual. The symptoms increase until the fourth day. At that period (although sometimes a day later) little red spots, just like flea-bites, begin to come out on the forehead and the rest of the face. These increase both in size and number, group themselves in clusters, and mark the face with largish red spots of different figures. These red spots are formed by small red papulae, thick set, and just raised above the level of the skin. The fact that they really protrude, can scarcely be determined by the eye. It can, however, be ascertained by feeling the surface with the fingers. From the face—where they first appear—these spots spread downwards to the breast and belly; afterwards to the thighs and legs. Upon all these parts, however, they appear as red marks only. There is no sensible protuberance by which they show themselves above the level of the skin.

News and Comment

UNIVERSITY NEWS

Dr. Borden S. Veeder, clinical professor of pediatrics at the Washington University Medical School, St. Louis, was granted the honorary degree of Doctor of Science by Colgate University recently.

Dr. Richard A. Bolt, assistant professor of child hygiene and lecturer in pediatrics at the University of California, has resigned to accept an appointment as director of the Cleveland Child Health Association, associate professor of hygiene and public health, Western Reserve University Cleveland, and associate in preventive pediatrics at the medical school.

Dr. Robert A. Black, professor of pediatrics at the Loyola University School of Medicine, Chicago, will have charge of the new $300,000 La Rabida Sanatorium in Jackson Park. The building will soon be under construction, and it will be used for the care of children with heart disease.

The University of California Medical School announces the following promotions: Dr. Ernest Wolff, assistant clinical professor of pediatrics; Dr. Clain F. Gelston, assistant clinical professor of pediatrics, and Dr. Edward B. Shaw, assistant clinical professor of pediatrics.

CLINICS

Sixty-six children attended the first behavior clinic of the New Hampshire state board of health, held at Durham. The purpose of this clinic is to assist parents to avoid bad mental habits in children.

GENERAL

Malarial Index Test.—Under cooperation of the state board of health, the county board of health and the U. S. Public Health Service, a malarial index test will be carried out on about 20,000 school children in Shelby County, Tennessee.

Lectures on Respiratory Diseases and Tuberculosis in Infancy and Childhood.—A series of lectures in English on "Respiratory Diseases and Tuberculosis in Infancy and Childhood" will be given by Dr. P. F. Armand-Delille from 10 to 12 a. m., Oct. 7 to 12, 1929, at the Hôpital Herold, place du Danube (XIX^{e}), Paris. The fee is 500 francs ($20). The course is given under the direction of the Association pour le developpement des relations medicales.

Bequests and Donations.—The following bequests and donations have been announced: Children's Homeopathic Hospital, and Home of the Merciful Savior for Crippled Children, Philadelphia, $24,000 from the estate of Miss Louisa C. Reisterer; Childrens Aid Society, New York, $10,000 by the will of Brander Matthews; Children's Hospital of Los Angeles, $100,000 by the will of Mrs. Grace Velie Harris; Babies Hospital, Wrightsville Sound, Washington, D. C., $10,000 by the will of Charlotte Arnold H. Bryson.

SOCIETY NEWS

Interstate Post Graduate Medical Association of North America.—The Interstate Post Graduate Medical Association of North America will meet in Detroit, from October 21 to 25. Among prominent pediatricians who will appear on the program are: Dr. Alan Brown, associate professor of medicine, Toronto; Dr. H. B. Cushing, professor of pediatrics, McGill University, Faculty of Medicine,

Montreal; Dr. John C. Gittings, professor of pediatrics, University of Pennsylvania School of Medicine, Philadelphia; Dr. McKim Marriott, professor of pediatrics, Washington University School of Medicine, St. Louis; Dr. Herbert B. Wilcox, professor of diseases of children, Columbia University College of Physicians and Surgeons, New York.

International Congress of Pediatrics.—An International Congress of Pediatrics will be held at Stockholm, Sweden, Aug. 17-20, 1930. Communications should be sent to Prof. I. Jundell, Artillerigatan 23, Stockholm, Sweden.

PERSONAL

Dr. Thomas P. Brennan, formerly acting director of the University of Iowa Psychopathic Hospital, has accepted a position as superintendent of the Wayne County Training School, Detroit, which handles problem children from the courts of that city.

DEATHS

Louis Guinon, prominent French pediatrician, died April 12, 1929, at the age of 69. He held important posts as chief of service at l'hôpital Trousseau and later at l'hôpital Bretonneau. Some of his more important contributions to pediatric literature are: Compression of the Trachea by Tuberculous Glands (1903); Relation of Air-Swallowing and Vomiting in Infants (1904); Interpretation of Roentgenograms of the Chest (1925). For many years he was contributing editor for both *Le Nourrisson* and the *Archives de medicine des enfants.* In 1899, he was one of the founders of the Societé de pediatrie. During the war he founded a tuberculosis dispensary which was later taken over by the government but still functions under the name "Dispensaire Louis Guinon." After the war the American Committee of the Argonne Association entrusted to him the organization and direction of a hospital for orphans. His reputation rested on his ability as a clinician, organizer and medical writer.

Carlo Francioni, director of the pediatric clinic at the University of Bologna, died on June 15, at the age of 52. He had been in charge of the clinic at the University of Bologna since 1915, and was responsible for the great advancement of that department. During this time and previously he had contributed many interesting treatises to pediatric literature.

Dr. Alice Outhouse of the Massachusetts General Hospital who, though quite young, had contributed materially to pediatric research, died in that hospital, on June 21, of meningitis.

Abstracts from Current Literature

Pathology, Bacteriology and Biochemistry

A Study of Certain Effects Occasioned in Dogs by Diphtheria Toxin. Harold J. Stewart, Arch. Path. **7**:767 (May) 1929.

Injection into dogs of 0.00135 cc. of diphtheria toxin per kilogram of body weight was followed by a decrease in the size of the heart. Checking factors which might be responsible for this revealed that there was no decrease in the number of red cells and no consistent change in the volume of blood; hence, destruction of blood was probably not a factor. The possibility of injury to the capillary bed was considered, but no evidence was found. No gross or microscopic lesions could be made out in the heart muscle, although there was a definite decrease in its weight. The reason for this was not evident.

Leslie, Evanston, Ill.

The Origin and Fate of Sickle-Shaped Red Blood Cells. Joseph Levy, Arch. Path. **7**:820 (May) 1929.

Fresh blood smears, sealed with petrolatum or Canada balsam, may be studied for a period of several days or more. Of 213 negro patients, there were 12 whose blood showed sickling. The sickling developed readily at room temperature and more rapidly at 37 C. Later change consisted in the fragmentation of the corpuscles. Refrigeration fixes the cell in its existing shape. The author thinks that the cell which develops sickling is a young cell which has just extruded its nucleus and is unable to assume the biconcave discoid form. Its frequent occurrence with nucleated red cells tends to substantiate this belief.

Leslie, Evanston, Ill.

The Action of Irradiated Ergosterol in the Rabbit. M. I. Smith, Pub. Health Rep. **44**:1245 (May 24) 1929.

The action of irradiated ergosterol was studied in the rabbit with especial reference to its toxicity and its influence on serum calcium, inorganic phosphate and tissue calcium. Repeated administration of 2 mg. and more of irradiated ergosterol given orally or intramuscularly to full-grown rabbits may prove fatal in a relatively short time. Doses of 1 mg. or less given three or four times a week seem to be well tolerated. Large doses produce a more or less marked hypercalcemia. Small doses produce variable results. The inorganic phosphate of the serum, though subject to considerable normal fluctuations, also appears to be definitely increased under the influence of large doses of irradiated ergosterol. Toxic doses produce great increases in the calcium of certain tissues. This calcifying action appears to be highly selective, involving certain tissues, e. g., the aorta, kidney and lungs, but not the liver or striated muscle.

Sanford, Chicago.

Digestive Enzymes of the Human Foetus. M. E. Lucas Keene and E. E. Hewer, Lancet **1**:767 (April 13) 1929.

The conclusions were based on a study of thirty-nine fetuses. Only material which was received within twelve hours of birth and which showed no autolytic changes at autopsy was used. The youngest fetus was 16 weeks old.

The authors concluded that:

1. Proteolytic ferments were present at 16 weeks.
2. Hydrochloric acid was present at 19 weeks.
3. Rennin was present at 19 weeks.
4. Pancreatic lipase was present at 32 weeks.
5. Carbohydrate splitting ferments were found rarely at 24 weeks and not constantly at full term.

Langmann, New York.

FUNCTION OF THE APPENDIX. J. SIEGL, Arch. f. Kinderh. **86**:171 (Feb. 22) 1929.

In a girl, aged 2½ years, Siegl made a diagnosis of pneumonia accompanied by symptoms of a pseudo-appendicitis. He then observed the function of the appendix by roentgen examination and found all the symptoms of appendicitis disappeared with the symptoms of the disease of the lung. In 75 per cent of all children with healthy intestines, it was observed that the appendix filled up with the contrast meal and emptied before or at the same time as the cecum and the adjoining parts of the ascending colon. In cases of appendicitis or other diseases of the cecal region, the appendix showed, besides fixations, distortions and displacements, an isolated permanent fulness which lasted after all other parts of the intestine had been emptied. If this evacuation became normal after the spontaneous recovery from pneumonia, the author considered it only a symptom of the latter. In the reported case of pneumonia, the first roentgenogram revealed a filling of the appendix that persisted for forty-eight hours after the evacuation of the intestine, which was functioning normally. Later investigations showed the same filling of the appendix, but its evacuation occurred at the same time as that of the intestine.

THE BIOLOGY OF THE VAGINA IN INFANTS. G. ABRAHAM, Arch. f. Kinderh. **86**:211 (Feb. 22) 1929.

Abraham studied the vaginal secretion of forty infants, from 1 month to 3 years of age, using the gasket method of Schroder and Keesler. He found that the younger the child the more the secretion tended toward neutral in the normal patient. For older children, the average p_H was 4.83. In pathologic conditions, such as both acute and chronic generalized infections, the p_H rises to definite alkalinity, the highest occurring in a case of meningitis when it rose to 7.65. The bacterial flora changed in accordance with the reaction. SANFORD, Chicago.

THREAD-FORMING PROPERTY AND STRUCTURE IN EGG WHITE. J. JOCHIMS, Biochem. Ztschr. **203**:242, 1928.

The property of being drawn out into threads is common to many biologic substances. In egg white, beating and coagulation bring out the fine fibrillar structure on which this property seems to depend. Human sputum is similar. A fibrillar structure has also been demonstrated for a number of other substances, for example, soap and hydrocellulose. LESLIE, Evanston.

REFINEMENT OF THE BENZIDINE TEST FOR BLOOD IN URINE. BOAS, Wien. klin. Wchnschr. **42**:368 (March 21) 1929.

Since salts and uric acid obscure the usual test, the author filters the urine, and applies the test to the residue. He can readily detect 1 part of blood in 200,000 parts of urine. CLAUSEN, Rochester, N. Y.

Nutrition

THE PROBLEM OF SWEETS FOR CHILDREN. H. C. SHERMAN, Child Health Bull. **5**:65 (May) 1929.

As sugar is almost devoid of proteins, vitamins and mineral elements, Sherman believes that it should be used cautiously. In general, the proper place of sugar in the food supplies and eating habits of children is not in such concentrated forms as candy, or in the sweetening of all kinds of foods, but rather as a preservative and flavor to facilitate the introduction of larger amounts of fruit and milk into the child's dietary. SANFORD, Chicago

DIARRHEAL CONDITIONS IN THE SOUTHERN STATES. EDWARD CLAY MITCHELL, South. M. J. **22**:35 (Jan.) 1929.

The article emphasizes the lack of a simple, uniform classification of diarrheal conditions in infants and the confusion that is prevalent among the general practitioners because of the many synonymous terms.

Hospital records are presented to show a typical classification and the mortality rate from diarrheal disturbances in Memphis.

The frequency of bacillary dysentery is also emphasized, especially where there is a bloody diarrhea. The author stresses the importance of laboratory work as a positive diagnostic aid in this type of disturbance. It may be added that bacillary dysentery due to the various strains of dysentery bacilli may not always be accompanied by bloody diarrhea.

SCHLUTZ, Minneapolis.

A CASE OF SEVERE RICKETS. NORMAN HILL, Proc. Roy. Soc. Med. **22**:758 (April) 1929.

A girl, aged 7 years, was admitted to the hospital because of twisted legs which kept her from walking with ease. Roentgenograms showed active rickets. The blood calcium amounted to 8.3 mg. and the inorganic phosphorus, 3 mg. Under treatment with cod-liver oil, a cod-liver oil concentrate, artificial sunlight and massage, the patient improved rapidly. Roentgenograms taken six months later showed marked improvement; the blood calcium went up to 10.2 mg. and the blood phosphorus to 5.8. The patient walked easily, although her movements were awkward owing to deformities.

WILLIAMSON, New Orleans.

THE TOXICITY OF IRRADIATED ERGOSTEROL. H. SIMONNET and G. TANRET, Presse méd. **29**:468 (April 10) 1929.

Doses of irradiated ergosterol amounting to from 500 to 5,000 times the active dose produced no toxic effect in the rat. For this reason, the authors do not believe that therapeutic doses of irradiated ergosterol could cause any accidents of hypervitaminosis in the infant.

GENERAL INDICATIONS FOR THE CHOICE OF FORMULAS FOR INFANTS. M. MICHALOWICZ, Rev. franç. de pédiat. **4**:608 (Oct.) 1928.

An interesting and simple classification of nutritional disturbances is presented by Michalowicz. He divides them into two classes: those concerned with the digestive tract alone and those concerned with the entire organism, that is, disturbance in assimilation and its consequences. Disturbances in both these realms may be due either to faulty feeding, to constitutional deficiencies of the organism or to both. These severe conditions, intoxication and decomposition, are always due to the latter cause. The thing that the author does not bring out is that improper feeding in itself may in time produce constitutional deficiencies.

When the food alone is at fault, he uses simple formulas of milk, water and carbohydrate. When the fault is in the organism itself, he emphasizes the importance of general hygienic and stimulative treatment in addition to the use of special foods such as breast milk and albumin milk.

LESLIE, Evanston.

A CASE OF INCIPIENT SCURVY. M. OLINTO, Arch. de pediat., Rio de Janiero **2**: 206 (March) 1929.

A child, aged 20 months, had anorexia, anemia, edema of the extremities and oliguria. The diet lacked vitamin C and was rich in farinaceous material. The dietetic treatment consisted of milk with 5 per cent carbohydrate, and lime juice. Scurvy is rare in Rio de Janeiro, Olinto having seen only two cases in the clinic in the past ten years. There may be many cases with slighter symptoms than the foregoing, and for this reason incipient scurvy should be looked for.

Weissenberg's Citric Acid Whole Milk as a Prophylactic Against Rickets. R. Behrens, Arch. f. Kinderh. **86**:90 (Jan. 25) 1929.

Behrens reports his results with acidulated unskimmed milk, which he gave to 109 children whose ages ranged from a few days to more than 5 months. The milk was tolerated well by all, especially the younger infants. The duration of this diet varied from four weeks to ten months. The weight increased quickly in most of the children; a few needed more time to adjust themselves. Symptoms of rickets found in some at the time of their arrival in the sanatorium not only were checked in their development but were diminished spontaneously. Among 60 children dismissed from the institution, 11 showed slight, 5 medium, and 8 serious symptoms of rickets. The author compared these results with his observations in the consultation center for infant care connected with his sanatorium and found, among 162 infants, 90 with no symptoms, 26 with slight symptoms, 25 with moderate symptoms and 21 with grave lesions. The author recommends the use of citric acid whole milk as it is a simple, inexpensive food, easily digested and prophylactic against rickets.

The Simulation of Pneumothorax by Thoracic Rickets. J. Siegl, Arch. f. Kinderh. **86**:173 (Feb. 22) 1929.

In a child, aged 18 months, with severe rickets, the head was enlarged and the fontanel was still open. The chest and ribs showed Harrison's groove and considerable flaring. A roentgenogram revealed the typical light area of a pneumothorax along the margin of the right lung. Siegl calls attention to the pathologic change shown by the form of the chest that may result from severe rickets and believes that this condition is more common than is ordinarily supposed.

Sanford, Chicago.

Treatment and Prevention of Rickets with Irradiated Ergosterol. P. Freud, Med. Klin. **25**:1709, 1928.

Irradiated ergosterol was given to fifty-seven children between the ages of 4 months and 2 years. Twenty-three of these children had active rickets before the beginning of the treatment.

Five drops of irradiated ergosterol were given twice a day for a period of from one to two and a half months, depending on the severity of the process. In all cases there was a complete clinical cure. The ergosterol was given for two days followed by an intermission of four days. The prophylaxis was continued from November to April. None of the children developed rickets, while four children under the same dietary regimen and in the same rooms who did not receive ergosterol showed active rickets. In two children, a smaller dose was given in the following way: Five drops twice a day for one day, followed by an intermission of five days. One of these children developed rickets. This seems to prove that the dosage was too small. In the children treated for rickets, active signs of improvement were seen after ten days of administration of irradiated ergosterol.

Zentay, St. Louis.

Rachitic Caput Natiforme. Ernst Podvinec, Monatschr. f. Kinderh. **43**:145 (April) 1929.

Before the time of the Wassermann reaction and the discovery of the organism of syphilis, the caput natiforme was considered an important diagnostic sign for the diagnosis of congenital syphilis.

In a careful study of six cases, the writer shows that typical caput natiforme can be produced by rickets. It is usually completely developed at about the end of the first year. It is interesting to note that the periosteal growth on the long bones is no greater in these children than in those with no caput natiforme. Caput quadratum, on the other hand, is not a definite sign of rickets, for the writer has seen it in children who were absolutely nonrachitic.

Gerstley, Chicago.

SALT-FREE DIET AND THE NEW SUBSTITUTE FOR SALT. F. O. HESZ, München. med. Wchnschr. **75**:570 (April 15) 1929.

Because salt is restricted or eliminated in a number of conditions, especially diseases of the kidney or the circulation, a substitute is essential to satisfy the taste of most patients. The author believes that a substitute of this kind has been found. The cations are sodium and calcium, and the anions are amino-acids (glutamic and formic). It contains no halogens and less sodium than salt. When added to food, it imparts the taste of salt. It is hoped to develop it into a "freely-pouring" substitute for salt.

SCHORER, Kansas City, Mo.

NUTRITIONAL DISTURBANCES IN INFANCY. R. WAGNER, Wien. klin. Wchnschr. **42**:395 (March 28) 1929.

A lecture in an extension course giving the current views on the subject does not lend itself to a brief abstract and contains nothing new.

CLAUSEN, Rochester, N. Y.

ALTERATIONS IN THE ACID BASE METABOLISM IN ALIMENTARY AND IN SODIUM CHLORIDE FEVER. L. SCHÖNTHAL, Ztschr. f. Kinderh. **46**:491 (Oct. 31) 1928.

The clinical picture of alimentary intoxication (anhydremia), after the exhibition of a food rich in calories, was observed in an infant when the water intake was greatly decreased. The alterations that then occurred in the acid base equilibrium were a great concentration of the plasma, a diminution of the bicarbonate and a big increase in the chloride concentration. In six others cases, a disturbance of the water economy was caused by the great quantity of salt in the food given. Here, also, in spite of the fever a disturbance of the metabolism occurred as manifested by acidosis. In two of the six cases, the attempt was made to set up a complete balance of the strong acids and bases of the blood and urine. Corresponding to a diminution of the plasma bicarbonate which would seem to point to a loss of alkali in the urine, an increase in bicarbonate excretion was discovered. The ammonia coefficient and György's total acid coefficient were correspondingly diminished. Further study showed the possible danger of the administration of great quantities of sodium chloride to infants who have had diarrhea and loss of water. The infusion of sodium chloride solution in such cases may lead to an increase of the acidosis and may hinder the restitution of the normal acid base equilibrium.

BASAL METABOLISM IN CHILDREN AND ADOLESCENTS. A. ROSENBLÜTH, Ztschr. f. Kinderh. **46**:531 (Oct. 31) 1928.

The figures for the basal metabolism of normal children published by Benedict and Talbot are too low for the normal cases reported here, but the figures given by Kistner and Knipping agree very well, most of the determined values, in comparison with the theoretical values, varying from minus 5 to plus 15 per cent. The predicted basal metabolism can be calculated approximately from the sitting height; the sitting height squared multiplied by 0.233 gives the predicted basal metabolism in calories. The figures, reckoned from the sitting height, agree approximately with those of Kistner and Knipping. The deviations from the predicted values are normally greater than with those for adults, and the frequency of the deviation from the zero line is especially great between the ages of 14 and 16. Between the constitutional types and values for basal metabolism, no definite regularity is found.

EMERSON, Boston.

Prenatal Conditions

THE EFFECTS OF X-RAY AND RADIUM UPON THE FETUS IN UTERO. PERCY W. TOOMBS, Am. J. Obst. & Gynec. **17**:516 (April) 1929.

The author has carefully reviewed the literature dealing with the experimental and clinical effect of the x-rays and radium on the fetus in utero. He differentiates definitely between the diagnostic and therapeutic use of roentgen exposure. He concludes that there is no evidence that the x-rays used in the usual diagnostic dose will affect the fetus in utero, but that there is a grave danger to the infant when therapeutic doses of the x-rays or radium are used during pregnancy. The results are largely dependent on the period of gestation. In the early months exposure usually results in abortion, while in the later months deformities, particularly of the central nervous system, are commonly the result. The younger the fetal tissues are when the dose is given, however, the more pronounced the injuries are. Toombs gives a timely warning against the therapeutic use of these agents in all gynecologic conditions, except malignant disease, when it is not known definitely that the patient is not pregnant.

ALLEN, Chicago.

CONGENITAL ATRESIA OF THE ESOPHAGUS WITH TRACHEO-ESOPHAGEAL FISTULA. W. LIKELY SIMPSON, Arch. Otolaryng. **9**:267 (March) 1929.

Cautley's classification of malformations of the esophagus is given as follows:

1. Complete absence, usually associated with gross malformation as in monsters.
2. Double esophagus, with reunion at lower end (rare).
3. Diverticula or pouches, pharyngeal rather than esophageal, situated posteriorly at the junction of the pharynx and esophagus.
4. Cysts.
5. Tracheo-esophageal fistula, without any other abnormality (rare).
6. Congenital dilatation, usually just above the diaphragm (quite rare).
7. Atresia without fistulous communication with the trachea, a malformation of various types and rather common.
8. Atresia with the lower end of the esophagus opening into the trachea and rarely into the bronchus. The majority of the cases are of this type and are remarkably alike in anatomic appearance.

Other structures of the body may be malformed with the last named type.

The symptoms are: The infant takes food which is immediately regurgitated; coughing, choking and cyanosis occur when it attempts to take food; tympanites is present if there is enough of an opening to force air into the stomach. Terminal starvation and bronchopneumonia are present if some other cause of death does not supervene. The x-rays may be used to verify the diagnosis, as may also the attempt to pass a catheter.

The course usually lasts only from five to eight days, though the author reports three cases, in one of which the patient lived for sixteen days, a gastrostomy having been done early. This is the longest duration of life in any reported case. Since all cases terminate fatally, treatment can only be directed at prolonging life. Gastrostomy is the only practical treatment.

BARNETT, Chicago.

DEVELOPMENT OF PREMATURE INFANTS IN FEEDING STATIONS. ILSE ZIMMERMANN, Monatschr. f. Kinderh. **43**:135 (April) 1929.

From a study of 107 cases in the poorest district of Vienna, the writer concludes that premature infants weighing 1,000 Gm. and more at birth can be taken care of just as well in the home as in an institution. The deciding factor is, of course, the problem of nutrition. If breast milk is available and if a continual nutritional disturbance, asphyxia, convulsions and subnormal temperature are not present, infants weighing from 1,000 to 1,500 Gm. will do well in the home.

The open feeding stations seem to be conducted like the infant welfare stations in America and have been successful in reducing infant mortality.

GERSTLEY, Chicago.

MONSTERS (ZRUDY). JOSEPH SAIDL, Sborn. lék. **29**:109, 1927.

The author presents the history, etiology, frequency and obstetric and surgical aspects of monsters. A complete anatomic classification of monsters is given. Photographs, roentgenograms and comprehensive descriptions of 105 specimens from the Hlava Institute of Pathological Anatomy are presented. A bibliography is included.

STULIK, Chicago.

Diseases of the New-Born

GRAVE FAMILIAL JAUNDICE OF THE NEWLY BORN. A. C. HAMPSON, Lancet **1**:429 (March 2) 1929.

Grave familial jaundice is described as a condition that occurs in successive infants of the same family. The jaundice usually shows itself on the first day and rapidly deepens. The liver and spleen, as a rule, are not enlarged at birth and often do not become enlarged during the course of the disease. The stools are normal in color.

Increasing jaundice, increasing drowsiness and loss of weight are the outstanding symptoms. Death usually occurs within three weeks, frequently with convulsions.

The hemolytic nature of the disease is supported by the fact that the infants become progressively anemic. The author believes that the accumulation of the toxic products of hemolysis inadequately excreted by the liver sufficiently account for the symptoms. Cases in which there is definite maternal toxemia appear to belong to a different group.

Points of differentiation between this disease and familial acholuric jaundice, congenital obliteration of the bile ducts, congenital syphilis and infective jaundice are cited.

The prognosis for untreated patients is poor; of ninety untreated patients, seventy-five died (83 per cent). Of eighteen treated patients seventeen recovered (the patient who died was first seen late in the course of the disease).

The treatment consists in giving mother's serum intramuscularly in daily doses (from 5 to 15 cc.) until the bilirubin content of the plasma has fallen to approximately 1 : 150,000. The total amount varied from 30 to 60 cc.

LANGMANN, New York.

GRIP ASSOCIATED WITH A MORBILLIFORM RASH AND A SEPTIC TERMINATION IN THE NEW-BORN. S. ROSENBAUM, Monatschr. f. Kinderh. **43**:153 (April) 1929.

Five new-born infants developed a morbilliform rash which was somewhat redder than measles. There was no conjunctivitis and no Koplick's spots, but a purulent nasopharyngitis was present. The disease ended in sepsis with fatality in four cases.

Against the diagnosis of measles is the age of the patients, the absence of Koplik's spots and conjunctivitis, and the fact that there were no other cases of measles in the institution. The rash did not have the appearance or the scaling of the erythema neonatorum toxicum of Leiner. In three cases, influenza bacilli were isolated from the mucous membranes.

The writer describes the condition as morbilliform influenza of the new-born, terminating in streptococcic sepsis.

GERSTLEY, Chicago.

BIRTH TRAUMAS OF THE CENTRAL NERVOUS SYSTEM. OTTO ULLRICH, München. med. Wchnschr. **76**:487 (March) 1929.

According to Lenz, health turns to disease when the coordination of an organism nears the limits beyond which life no longer is possible.

Usually only the trauma that results from long labor (due to disproportion of the fetus and the female organs and parts concerned in birth) is considered, but even in normal labor the skull containing the brain must be compressed in order

to be forced through the birth canal. Due to the small outlet, atmospheric pressure varies between the born and the unborn parts of the fetus. So long as the membrane has not ruptured, the fetus itself need not bear the immediate brunt. When it has ruptured, however, the unborn portion is first under pressure; then, as more of the fetus is born, a negative pressure is produced. In this way, the fontanels and therefore the brain are under negative pressure or under pressure causing leakage from the blood vessels of and surrounding the brain, especially the meningeal vessels and the vena magna galeni. Schwartz believes that the negative pressure is the most important. Breach births result in more damage than cephalic births.

That most persons are born without damage is their good fortune. Of all conceptions, however, 3.5 per cent terminate in stillbirths, and 3.5 per cent result in death during the first week of life. Of 1,300,000 infants born in 1925, 80,000 died in the first month; 40,000 of these were premature.

In the premature infants, Ylppo found hemorrhage in the brain and spinal cord almost exclusively. These children, even though they are small and are not so subjected to trauma, have less resistant blood vessels. This need not lead to profuse hemorrhage but only to punctate hemorrhages and softening of the nervous tissue. In addition, many lesions found later in life at autopsy that are regarded as secondary manifestations are probably due to hemorrhage at birth.

Sharp found bleeding in 10 per cent of the new-born infants; Ullrich found the percentage to be 15 in 100 cases of more severe labor. He believes that at least 10 per cent of all births are complicated by injuries at birth. Forceps deliveries have shown fewer hemorrhages.

Of the nonfatal hemorrhages much is to be learned. Dollinger ascribes to them many progressive nervous diseases and disturbances of the vegetative system. But here the author cautions one to be careful to differentiate this condition from the acute natal influences. Of the 10 per cent of the children in whom the author found blood in the spinal fluid, three died and the rest appeared normal at 5 years. Of children with definite clinical symptoms with cerebral hemorrhages, twenty died during the first week; of the eight that are living, two are idiots with spastic symptoms and six are normal.

One must not view too pessimistically the summary, which states that 3 per cent of stillbirths are due to birth trauma, that 10 per cent of infants born alive have blood in the spinal fluid and that 33 per cent of these die, 3 per cent have defects and 64 per cent show no defects, and that a large percentage have brain lesions which cannot definitely be traced to injuries at birth. One half of the stillborn infants are premature, primarily injured or deficient, and birth trauma may be a selective factor of value to the race. SCHORER, Kansas City, Mo.

MENINGITIS IN NEW-BORN INFANTS. Ö. NACHTNEBEL, Orvosi hetil. **73**:80 (Jan. 26) 1929.

Meningitis was found at autopsy in five new-born infants. Cultures showed pneumococci. The pneumococci were of type II in three cases and of type III in two. The ages of the new-born infants varied between 18 hours and 10½ days. The infection probably occurred in intra-uterine life during or before birth. The probable portal of infection was in the pharynx. The observations at autopsy were not characteristic. There was hardly any exudate, and the diagnosis could be made only through bacteriologic and histologic examination. ZENTAY, St. Louis.

Acute Infections

EPIDEMIOLOGY OF WHOOPING COUGH. EDWARD S. GODFREY, New York State J. Med. **28**:1410 (Dec. 1) 1928.

A study of the statistics for the United States and New York state shows interesting epidemiologic data. Although whooping cough is supposed to be transmitted by secretion from the respiratory tract, it does not show the decline during

the summer characteristic of other diseases thus transmitted. While the highest mortality is found in March, there is a sharp rise in August.

The death rate is higher in rural than in urban communities throughout the United States, except in New York, New Jersey and the New England states. The mortality among the negroes in the South is higher than that of the white race. In New York state, the lowest mortality has been in villages of less than 2,500. The death rate for whooping cough is higher in southern than in northern states, this fact not being wholly accounted for by the greater mortality among negroes. Whooping cough shows a preference for females. In New York state, exclusive of cities with a population of more than 200,000, there were 112 deaths among females for every 100 deaths among males. Case fatality rates are unreliable because, according to the author, whooping cough is the most poorly reported lethal communicable disease of childhood. Isolation, especially of small infants, as well as large doses of freshly prepared vaccine is strongly advised.

THE EPIDEMIOLOGY OF AN OUTBREAK OF ILLNESS DUE TO MILK. FRANK W. LAIDLAW, New York State J. Med. **28**:1465 (Dec. 15) 1928.

Of eighty-two children and one teacher who were served with "Raw Grade A" milk, seventy children and the teacher were taken severely ill about three hours later. The symptoms shown were severe abdominal pain followed by vomiting and marked prostration. In some cases, diarrhea occurred. The children lost, on an average, two and one-half days of school. Thirteen other cases occurred outside of school; no deaths were reported. A careful investigation showed that the milk was infected by one of forty-two cows. Chronic mastitis affected one fourth of the animals; the other three fourths were comparatively normal.

A theory that the milk had been poisoned is still cherished by some of the people of that community.

AIKMAN, Rochester, N. Y.

CHICKENPOX AND WHOOPING COUGH: PREVENTION AND TREATMENT. ROY M. GREENTHAL, Wisconsin M. J. **27**:540 (Dec.) 1928.

In combating epidemics of chickenpox, the author reports success both by vaccination with the contents of fresh vesicles and by the use of convalescent serum. Protection from vaccination lasts about one year and from serum, fifty days. A combination of the two methods is advised in epidemics in hospitals. No recent therapy, including vaccine, roentgen, ultraviolet ray and alkali therapies, has given satisfactory results in whooping cough.

MEASLES AND THE COMMON COLD. M. G. PETERMAN, Wisconsin M. J. **27**:540 (Dec.) 1928.

Measles may be efficiently prevented if convalescent serum, convalescent whole blood or Tunnicliffe's antimeasles immune goat or horse serum is used in sufficient amount during the first four days after exposure. The passive immunity derived lasts from six to ten weeks. Of seventy-four patients exposed, 91 per cent failed to develop measles after immunization with the antimeasles immune goat and horse serum. From 5 to 10 cc. was used.

The common cold is a most serious yet neglected pediatric problem. The etiology is unknown and the treatment symptomatic. The pathologic condition is often slight yet may produce severe symptoms. It is not proved that a cold follows contact exposure, chilling and dampness or fatigue. Probably a combination of factors unite to favor its development. Vaccine has been demonstrated to be of no value.

SCARLET FEVER IMMUNIZATION. M. R. FRENCH, Wisconsin M. J. **27**:543 (Dec.) 1928.

Great variation in the potency of different preparations of Dick serum was demonstrated by retesting and by using two different preparations simultaneously. In prevention and treatment, convalescent serum has been difficult to obtain and

the prophylactic antitoxin has given severe reactions with protection of from only two to three weeks. Scarlet fever toxin changed from 66 to 85 per cent of the reactions to the Dick test from positive to negative. Larson's antigen gave similar results. About 30 per cent of these patients who had given negative reactions to the Dick test gave positive reactions in one year.

IMMUNIZATION FROM THE PUBLIC HEALTH STANDPOINT. W. W. BAUER, Wisconsin M. J. **27**:545 (Dec.) 1928.

Immunization is the greatest factor in the eradication of contagious disease. Smallpox, diphtheria and, under certain conditions, typhoid fever are, however, the only diseases in which prevalance and dependable results from immunization indicate unrestricted measures. The health department is considered as the logical unit to do this work. In measles, immunization may be recommended under some conditions. The attitude of the health department toward immunization and therapeutic procedures of doubtful value must be wholly conservative. No serum or vaccine procedure is advised in pertussis or scarlet fever.

DISCUSSION ON CONTAGIOUS DISEASES. ARCHIBALD HOYNE, Wisconsin M. J. **27**:548 (Dec.) 1928.

Antimeasles goat serum has proved of the greatest value as a prophylactic in institutional epidemics. Pertussis vaccine seems to prevent pertussis occasionally. Most deaths from diphtheria are due to errors in diagnosis. The family physician is inclined to postpone immunization with toxin-antitoxin because of the fear of sensitizing the patient. Active immunization against scarlet fever by use of the toxin seems of value. The antitoxin for passive immunity seems harmful rather than helpful. In severe cases, convalescent serum is effective.

JANNEY, Milwaukee.

THE ILLNESS OF INFECTION IN MEASLES. G. F. ABERCROMBIE, Brit. J. Child. Dis. **26**:15 (Jan.-March) 1929.

The author substantiates the observations of E. W. Goodall and cites several instances in which, on the occurrence of a case of measles, he examined other members of the family who had been in contact and found evidence of "naso-pharyngeal or respiratory catarrh" with the development of measles about twelve days later. If such patients are immediately isolated, the spread of the disease may be diminished. He also suggests that those who show "an illness of infection" would appear to be ideal subjects for treatment with measles serum.

A CASE OF TETANUS SIMULATING TETANY. LESLIE COLE, Brit. J. Child. Dis. **26**:26 (Jan.-March) 1929.

A boy, aged 2¾ years, was admitted to the hospital with a history of a graze on the left elbow fourteen days before and double otitis media of four days' standing. There was spasm of the masseter muscles, and the Chvostek and Trousseau signs were not elicited. On lumbar puncture, intense tonic spasm of the muscles of the face, jaw, neck, larynx and limbs resulted. The possibility of tetany was only suggested by the nature of the spasms involving the muscles of the extremities.

The child died shortly after admission, not responding to tetanus antitoxin intrathecally or intramuscularly or to any of the other measures tried.

LANGMANN New York.

HEMIPLEGIA AFTER DIPHTHERIA. D. W. WINNICOTT, Proc. Roy. Soc. Med. **22**:392 (Feb.) 1929.

A girl, who was healthy except for a left hemiplegia, at the age of 8 months had caught nasal diphtheria from her brother and was in the hospital for six weeks, but did not receive any antitoxin. When the mother took her from the

hospital she noticed that the child's face was weak on the left side, and that she avoided using her left hand. The patient walked at 2 years but with weakness in the left leg. The degree of paresis diminished steadily after the onset.

WILLIAMSON, New Orleans.

PROTECTION AGAINST SMALLPOX IN FRANCE. L. CAMUS, Presse méd. **40**:649 (May 18) 1929.

France depends more on the establishment of so-called "sanitary frontiers" at her borders than on internal procedures. A strict watch is kept for entering travelers who might themselves be infected, or carriers in any way. If there is any doubt, detention is made at the entering port until the period of quarantine is over, or until vaccination has been performed. Frequent revaccination is insisted on.

SYMPTOMATIC PURPURA HEMORRHAGICA AFTER RUBELLA. T. PITTEN, Arch. f. Kinderh. **86**:114 (Jan. 25) 1929.

The case is reported of a child, aged 9 years, with bleeding from the nose and mouth, following German measles eight days before. Her body was covered with petechial hemorrhages. The blood showed 4,090,000 red cells, 5,600 leukocytes and 120,000 platelets. The coagulation time was normal, but the bleeding time was thirty-two minutes. The blood calcium was 13.8 mg. A week later, the petechial spots had disappeared and the platelets had risen to 220,000. Four months later, the platelets were 270,000. Pitten reviews the literature regarding the relation between symptomatic purpura and the various diseases with which it is associated.

INVESTIGATIONS OF DIPHTHERIA CARRIERS. A. KOLLMANN, Arch. f. Kinderh. **86**:185 (Feb. 22) 1929.

Kollmann reports statistics on diphtheria carriers. Among 3,062 children admitted in the period from 1920 to 1928, 418, or 13.6 per cent, were diphtheria carriers. Younger infants most frequently are carriers. In autumn, winter and spring, the number increased. Unhygienic housing conditions produced a high percentage of diphtheria carriers. Children with digestive disturbances, exudates or nasopharyngeal diseases were affected particularly often. The increase of diphtheria bacilli was followed by an increase of morbidity within one month, while a decrease in the number of carriers was followed after about a month by a decrease in the cases of diphtheria. Thirty-eight per cent of new-born infants are carriers. The best treatment was found to be isolation of the carriers and the application of 2 per cent solution of silver nitrate.

SANFORD, Chicago.

SCARLET FEVER ANTISERUM. W. STOTTENBERG, München. med. Wchnschr. **76**:360 (March 1) 1929.

In the last one and one-half years the author had under his care about 800 children with scarlet fever; 153 of the sicker children received the antiscarlet fever serum. Of these, 66 showed a prompt reduction in temperature, 21 a gradual reduction, 14 no certain result, 20 a sharp rise of temperature after the initial drop, 32 no effects and 6 patients died. Thus, 87 were benefited and 66 showed little or no benefit. Of 66 patients who received serum early, 30 per cent had complications.

The author concludes that the serum has little influence on complications, and is of no value for relief from angina, otitis and adenitis. It is contraindicated in the rheumatism accompanying scarlet fever, and except in the highly toxic cases he does not advise its use.

SCARLET FEVER. H. SCHOTTMÜLLER, München. med. Wchnschr. **76**:615 and 670 (April 12 and 19) 1929.

After attending the German-Russian Scarlet Fever Congress held this year, Schottmüller presents his views concerning the disease. At the congress the question, "What is scarlet fever?" was not answered; the author says that he cannot give the generally accepted answer.

The reason for the uncertainty lies in the fact that knowledge of the etiology is incomplete. The views of the etiology may be divided into four groups: (1) an unknown virus, (2) a hemolytic streptococcus which in addition to pyogenic functions may with variations produce scarlet fever, (3) sensitization or allergy produced by one streptococcus or its toxin so that the second streptococcus can produce the disease and (4) a particular scarlet fever hemolytic streptococcus.

All authors agree that the contagious material is highly specific and that streptococci prevail or are present in the disease, that streptococci usually produce no immunity and that after recovery from scarlet fever there is immunity. Schottmüller regards the following facts as good arguments for the acceptance of a "scarlet fever streptococcus"; the finding of streptococci, by the Dick and Dochez methods, that produce a toxin which causes a reaction in the susceptible person and none in the convalescent; the fading of the exanthem after the injection of immune serum, and the reduction in fever observed when injections of antiscarlet fever serum are made.

Basing his conclusions on the results from the use of serum in the treatment of 736 of 1,000 patients, Schottmüller stated: (1) Seventy per cent of patients with cerebral symptoms were saved; (2) the fever was rapidly reduced; (3) complications occurred with about the same frequency; (4) serum diseases with symptoms in the joints occurred in 28 per cent of the cases, and (5) in a severe case of scarlet fever he would prefer to inject the blood from a patient convalescing after scarlet fever to injecting that from immunized horses.

He believes that the antiserum from horses or convalescent patients kills the streptococci quickly and certainly, due to a physicochemical rather than a bactericidal action. He contends that even if the problem of scarlet fever is not solved, there is a scarlet fever toxin, a serum of value in treatment and a method of active immunization.

SCHORER, Kansas City, Mo.

THE TREATMENT OF MALARIA WITH PLASMOCHIN. B. NISSENBAUM, Wien. klin. Wchnschr. **42**:300 (March 7) 1929.

Plasmochin has given excellent results in the treatment for quartan and tertian malaria. Relapses are much less frequent than after the administration of quinine. In tropical malaria, this preparation should always be given in conjunction with quinine. It destroys the gametes of tropical malaria. In cases of idiosyncrasy toward quinine, in conditions not amenable to quinine, in pregnancy and possibly in blackwater fevers, it may be substituted for quinine.

DIPHTHERIA BACILLI IN THE MALE URETHRA. J. WENDLBERGER, Wien. klin. Wchnschr. **42**:368 (March 21) 1929.

Although diphtheria bacilli have long been known as a cause of vulvovaginitis in young girls, the author has been unable to find reference to their occurrence in the male. He cites three cases of his own, and describes one in detail. The urethritis was of long duration, and the patient was nearly symptom-free. Large numbers of pseudodiphtheria bacilli accompanied the true ones, which appeared to be virulent for guinea-pigs, and which were not isolated in pure culture.

CLAUSEN, Rochester, N. Y.

A Case of Encephalitis After Vaccination. R. Priesel, Ztschr. f. Kinderh. **46**:569 (Oct. 31) 1928.

There can be no doubt that the case reported is of encephalitis or, more properly, encephalomyelitis. The remains of a recent vaccination could be seen; this was the first vaccination in the patient, a boy, aged 10 years, in whom at the typical time between the tenth and fifteenth day after vaccination, symptoms of disease of the central nervous system developed. This child exhibited perseveration and a much retarded perception; he made attempts to speak, brought out only unintelligible words, began to cry and turned away. His strength was so diminished that he could not get on his knees or his feet, except by the help of his arms, and he could not stand. According to most observers, encephalitis occurring after vaccination has only two courses: death or a complete cure. In the present case there was restoration to perfect health; the author reexamined the boy two years later, finding him sound even to the absence of transient pains of migraine-like character in the head. There were disturbances of the bladder, which have been reported several times in the literature in the disease picture of postvaccination encephalitis. Complete retention of urine was also present in this case.

Emerson, Boston.

Hemolytic Streptococcus and Scarlet Fever. L. Hornyánszky, Orvosi hetil. **73**:67 (Jan. 19) 1929.

In a newly opened ward, the nursing personnel of which previously worked in a scarlet fever ward, an unusually extensive epidemic of tonsillitis was noted. All the children gave pure cultures of hemolytic streptococcus. The search for a possible carrier resulted in finding pure cultures of hemolytic streptococci in the throat of one of the nurses. After isolation and treatment of this nurse, the epidemic promptly subsided.

At the beginning and at the end of the epidemic, the Dick test was performed on all the children. At the end of the epidemic, there was a considerable reduction in the number of positive reactions. The author thinks that the tonsillitis was a mild form of scarlet fever produced by hemolytic streptococci. This seems to be another proof of the specific action of hemolytic streptococcus in the etiology of scarlet fever. Isolation of patients with scarlet fever should be continued until the streptococci disappear from the throat.

Influence of Lipoids on the Action of Diphtheria Toxin in Animal Experiments. G. König and Z. Gulácsy, Orvosi hetil. **73**:90 (Jan. 26) 1929.

Cholesterin and lecithin were used in these experiments in the prevention and treatment of the effect of diphtheria toxin on guinea-pigs. In the prophylactic experiments, the lipoid was injected one and a half hours preceding the injections of the toxin. In the therapeutic experiments, the injections of lipoid were given three hours after those of the toxin. In the prophylactic experiments, injection of the lethal dose of toxin affected the guinea-pigs as follows: 0.02 Gm. of lecithin given subcutaneously prolonged life twenty-nine hours; 0.05 Gm. of cholesterin in oil prolonged it fourteen hours and 0.02 Gm. of colloidal cholesterin given intracardially prolonged it twenty-one hours. In the therapeutic experiments, a similar dose of lecithin prolonged life thirty-two hours, and cholesterin forty-two hours.

Hemorrhagic Preexanthems: A New Sign in Measles. G. Petenyi, Orvosi hetil. **73**:93, (Jan. 26) 1929.

In the prodromal stage of measles, small punctate hemorrhages can be seen on the oral mucous membranes. They can be found on the buccal membrane at about the same place where Koplik's spots are localized, and also on the soft palate. Their number varies between three and twenty. They usually appear from

one to three days before Koplik's spots can be seen, and last for about two or three days. Their diagnostic importance does not seem to be great, as they can be seen in many other conditions.

ZENTAY, St. Louis.

Chronic Infections

CONGENITAL LUES IN CHILDREN: ITS TREATMENT BY THE INTRAPERITONEAL INJECTIONS OF NEOSALVARSAN AND MERCUROSAL. JOSEPH YAMPOLSKY, Am. J. Syph. **8**:121 (Jan.) 1929.

The treatment of congenital syphilis by means of intraperitoneal injections of neoarsphenamine and mercurosal was attempted by the author after a series of animal experiments which made it appear to be a safe method to follow.

The results were as favorable as those obtained by the intravenous or intramuscular route. The immediate discomfort was considerably less in this method of administration.

STANDARDIZATION OF THE WASSERMANN TEST. RUTH GILBERT and VIRGINIA LANGWORTHY, Am. J. Syph. **8**:127 (Jan.) 1929.

The authors review the literature relevant to the relation between diabetes and syphilis. They conclude that there is little evidence that syphilis is a marked factor in the production of diabetes. They found, however, that in a few cases the diabetes was dependent on the syphilitic infection, and they conclude that when syphilis and diabetes are found in the same patient, their simultaneous occurrence is more frequently incidental than a matter of cause and effect.

They feel that a patient with diabetes, who also has syphilis, may be more prone to gangrene than the nonsyphilitic patient with diabetes, because of the syphilitic involvement of the vascular system.

MCBRIDE, Sioux City, Ia.

A STUDY OF THE TUBERCULOUS LESIONS IN THE LUNG OF A NEGRO CHILD NINE WEEKS OLD. WILLIAM SNOW MILLER, Am. Rev. Tuberc. **19**:119 (Feb.) 1929.

By means of serial sections and reconstruction, a careful study was made of a small tuberculous lymph node situated in the pleura, forming part of the boundary of an incisura interlobaris, together with its afferent and efferent lymphatics. Only in that portion of the lymph node which the lymphatic conveying bacilli supplied were tubercles developed. The other lymphatics that entered the node formed a wide open bypath through it. The toxins contained in the bypath and the associated sinuses gave rise to intense hyperplasia of the lymphoid tissue in the remainder of the node. The lymph flow, as shown by the presence of numerous valves, was toward the pleura. This showed that the direction of the lymph flow described for other portions of the lung holds true for this situation; namely, that from a narrow zone around the periphery of the lung the flow is toward the pleura, while in other portions of the lung it is toward the hilum. Beyond the lymph node, the flow was through the pleural network of lymphatics to a hilum node which showed active lesions. Lymphatics from other parts of the lung were seen entering the hilum node, showing that those described were not the only source from which it was infected.

SMITH, Ogden, Utah.

A REPORT OF THE STUDY OF 25,048 SCHOOL CHILDREN FOR TUBERCULOSIS. P. P. MCCAIN, South. M. J. **22**:310 (April) 1929.

In the series of school children reported in North Carolina, it was found that 22 per cent of the white and 27 per cent of the colored children reacted positively to the intracutaneous test for tuberculosis. Only 1.72 per cent of those having a positive tuberculin test had demonstrable tuberculosis.

Roentgenograms are presented illustrating what is considered to be the primary focus. The author confirms Ghon's statement that the primary focus is generally first present in the periphery of the lung, and that the resulting tracheobronchial tuberculosis is a secondary involvement. If the condition can be diagnosed at this stage, which is in all probability noncontagious, it is readily cured, especially in children more than 2 years of age.

SCHLUTZ, Minneapolis.

THE MUSCULAR ALTERATIONS OBSERVED IN THE SYPHILITIC OSTEOPATHIES OF INFANCY. M. PEHU and MADAME CLAVEL, Rev. franç. de pédiat. **4**:561 (Oct.) 1928.

Attention is called to the muscular changes in congenital syphilis engendered by an extension of the inflammatory process from the bone and periosteum or dependent on a syphilitic arteritis. Diminution in activity and the attitudes assumed by these infants are in a measure due to the muscular changes. The skeletal lesions alone are often insufficient to account for the pseudoparalysis.

LESLIE, Evanston.

ANTITUBERCULOSIS VACCINATION OF INFANTS WITH B. C. G. B. WEILL-HALLE and R. TURPIN, Rev. de la tuberc. **10**:9 (Feb.) 1929.

Since 1922, Weill-Halle, Turpin and their associates have been making a carefully controlled investigation of the value of B. C. G. as a preventive measure against tuberculosis. Special clinics were established for treatment and regular examinations, and a corps of nurses visited the homes. Among 152 vaccinated infants living in infected surroundings the mortality rate for the first year of life was 12.5 per cent, of which 3.9 per cent was possibly due to tuberculosis. After the first year, but one death occurred in this group. Among 392 vaccinated infants living in healthy surroundings, the mortality during the first year was 4.33 per cent, of which 1.02 per cent was presumably from tuberculosis. After the first year the mortality declined to 1 per cent during the second year and to 0.82 per cent during the third year. After the third year there were no deaths. Not any of this mortality was due to tuberculous infection.

From these studies covering a period of seven years, the authors draw the following conclusions: Vaccination with B. C. G. practiced on subjects not previously infected with tuberculosis is certainly inoffensive. It is effective when given by way of the digestive tract during the first ten days after birth, and after this period, when administered subcutaneously under the conditions indicated below. By mouth, three doses of 1 cg. each are given during the first ten days succeeding birth, with revaccination by ingestion at 1 and 3 years of age. Subcutaneous vaccination consists of one dose of from 0.01 to 0.02 mg. A period of observation of one month with two negative skin tests at intervals of eight days should precede vaccination by subcutaneous injection. Vaccinated subjects should be isolated temporarily to avoid infection during the period of immunization. Vaccination should be practiced on all infants likely to become infected with tuberculosis, on all whose parents seek vaccination, and on adults whose employment and surroundings invite infection.

MORTALITY IN INFANTS WITH FAMILIAL EXPOSURE TO TUBERCULOSIS. JACQUES PARISOT and SOLEUR, Rev. de la tuberc. **10**:45 (Feb.) 1929.

Since the institution of social hygienic measures and the prophylactic use of B. C. G., the mortality during the first year of life among infants with familial exposure to tuberculosis has abruptly fallen from an average of 18 per hundred to 10 per hundred. The average general infant mortality in this region for the same period was 11.3 per hundred. The value of the prophylactic measures used is attested by this reduction in mortality in those infants intimately exposed to tuberculous infection to slightly below the general average for the community.

A Bacteriologic and Experimental Study of Pus from Abscesses Following Injections of B. C. G. J. Parisot; L. Fernier, and H. Saleur, Rev. de la tuberc. **10**:50 (Feb.) 1929.

The subcutaneous injection of 0.01 mg. of B. C. G. is followed in about 5 per cent of the cases by a local reaction with the formation of a small abscess. Evacuation of the abscess by puncture results in rapid recovery. Examination of the pus from such abscesses reveals the presence of acid-alcohol resistant bacilli, to the exclusion of all other microbes. These bacilli, reinjected into an animal susceptible to Koch's bacillus, are incapable of causing the development of tuberculous lesions. The bacillus B. C. G., after a sojourn of from two to four months in the human organism, is incapable of recovering its tuberculogenic properties.

Smith, Ogden, Utah.

Smears from the Larynx. H. Edel, Med. Klin. **25**:1622, 1928.

Sputum taken directly from the larynx discloses acid-fast bacilli better because it is not mixed with mucus and saliva from the higher parts of the respiratory tract. The method is simple and is carried out by indirect laryngoscopy. The vocal cords, which are not anesthetized, are touched with a swab. This produces a violent attack of coughing, and the sputum thus obtained is taken directly with the swab and transferred on a slide.

The results are satisfactory, in the author's experience. In 500 cases, the positive results always corresponded with positive data obtained in sputum brought up spontaneously. In sixty-three cases he could find acid-fast bacilli in smears from the larynx when examination of sputum from spontaneous expectoration gave negative results.

Zentay, St. Louis.

The Malaria Treatment for Congenital Syphilis. H. Koch, Wien. klin. Wchnschr. **42**:400 (March 28) 1929.

Four children without symptoms of syphilis but with positive Wassermann reactions of the blood and the spinal fluid were treated with arsphenamine in conjunction with malarial inoculation. The malaria ceased spontaneously after from seven to nine paroxysms. The serologic changes disappeared, with the exception of a positive Meinicke reaction in one case.

Clausen, Rochester, N. Y.

Lactic Acid Content and p_H of the Cerebrospinal Fluid in Tuberculous Meningitis. J. Geldrich, Orvosi hetil. **78**:283 (March 23) 1929.

In tuberculous meningitis, the concentration of dextrose in the cerebrospinal fluid is decreased. This is accompanied by a proportionate increase of the content in lactic acid. Apparently, the decrease of sugar is due to its quantitative transformation into lactic acid. For diagnosis, determinations of lactic acid can take the place of the determination of dextrose. The chemical reaction is gradually becoming more acid and may reach a p_H of 7.22.

Zentay, St. Louis.

Internal Diseases

Bone Diseases in Infancy and Childhood. E. L. Jenkinson and Philip Lewin, Am. J. Roentgenol. **17**:201 (Feb.) 1927.

The authors present a table for the differential diagnosis of rickets, scurvy, syphilis, tuberculosis, nonspecific epiphysitis, osteochondritis, osteomyelitis, osteogenesis imperfecta, achondroplasia, Still's disease, marble bones, cretinism, multiple cartilaginous exostoses, multiple myeloma, coxa vara, hemophilia, coccidioidal granuloma, tumors, enchondroma, typhoid and typhus fever, infectious periostitis, aseptic necrosis and infantile osteomalacia. These conditions are discussed under the following headings: history, age incidence, changes in the various structures

such as the epiphyses, the epiphyseal line, the diaphyseal line, the shaft, medulla, cortex, periosteum, the joint, atrophy, fractures, deformities, treatment and prognosis.

THE RATIONALE OF RADIOTHERAPY IN HODGKIN'S DISEASE AND LYMPHOSARCOMA. ARTHUR U. DESJARDINS, Am. J. Roentgenol. **17**:232 (Feb.) 1927.

Radiotherapy exerts a strikingly beneficial influence on the lymphatic hyperplasia incident to lymphosarcoma and Hodgkin's disease, and the symptoms of adenopathy are relieved more or less completely for long or short periods, depending on the acute or chronic nature of the disease, on the stage reached when treatment is started and on the cooperation of the patient. At the outset it is best not only to treat the palpably enlarged nodes or masses of nodes but to give fairly general treatment of the nodes, especially those of the mediastinum and the lower paravertebral lymph chains. Four cases, with a diagrammatic outline of treatment, x-ray pictures, after-treatment and comment, are presented.

STULIK, Chicago.

DIGITALIS THERAPY—ITS USES IN CHILDREN. PHILLIP E. ROTHMAN, California & West. Med. **30**:150 (March) 1929.

Digitalis has fallen into disrepute among many pediatricians because it has been used in a wide variety of diseases without any rational pharmacologic basis. Experimental evidence shows that digitalis diminishes cardiac output, and hence diminishes cardiac work. In this sense, it is a cardiac sedative and not a cardiac tonic. This is one explanation of the beneficial action of the drug in heart failure associated with increased cardiac output. Digitalis is indicated only in cases of true failure of the heart muscle and not in acute circulatory failure or shock without evidence of visceral or peripheral congestion. Auricular fibrillation, a condition which responds most readily to the drug, rarely occurs in children. The chief indication for digitalis in children is heart failure due to acute rheumatic fever. Digitalis may be used to slow a rapid, regular, persistent heart action associated with rheumatic fever not due to fatigue, fever or excitement. The drug is ineffective when used to slow the heart rate in the acute infections accompanied by fever, in the cardiac neuroses, thyroid disturbances, acrodynia and paroxysmal tachycardia. It is never indicated in nephritis with edema, unassociated with heart failure.

The amount of the drug necessary for digitalization of children is relatively much greater than that for adults, although a marked individual variation exists. There is nothing better for oral administration than the powdered leaf or a tincture of high grade. The method of Jacobsen and Davison is satisfactory for digitalization.

Just when to stop the use of digitalis requires experience and judgment. The fear of producing digitalis intoxication is often responsible for stopping its administration before the effective dose is reached. It is better to give a little too much than not enough, as the effects of a mild intoxication are nearly always transient and readily subside with proper care. The distance, moreover, between a mild and a fatal intoxication is great. If signs of intoxication appear, the drug must be stopped until they have completely disappeared. The more severe the intoxication, the slower the elimination. The author emphasizes the importance of constant bedside attendance and observation during digitalis therapy for children.

JEIDELL, Salt Lake City.

NUTRITIONAL ANEMIA IN INFANCY: SOME OBSERVATIONS ON A COMMON DEFICIENCY DISEASE. HELEN M. M. MACKAY, Proc. Roy. Soc. Med. **22**:385 (Feb.) 1929.

The prevalence of anemia among infants in London was shown by an investigation extending over three years and comprising about 1,770 cases, with 3,100

examinations of the hemoglobin. The anemia was largely nutritional in origin and was due to a deficiency of iron, possibly associated with other factors. An infant obtains its necessary iron from milk, which is poor in iron, as well as from a store in the liver. The average percentage of hemoglobin in the blood of fairly healthy artificially fed infants in London shows a sharp fall from its high level at birth to about 65 per cent at 2 or 3 months of age, a rise to about 70 per cent by 5 or 6 months, then a fall again to 65 per cent at 12 months. The administration of iron, begun when the infant is 2 months of age, raised the hemoglobin to 80 per cent by 4 months. Infants whose weight at birth is under 6 pounds (2,727.5 Gm.), twins and premature infants are especially in need of iron. Iron and ammonium citrate proved effective and was used during the investigations, incorporated in a dried milk. No evidence was obtained that want of light is an etiologic factor in anemia in infancy; artificial light therapy did not cure the condition. No deficiency in vitamins appeared to be involved. The value of liver is still under investigation. The routine administration of an iron salt to artificially fed infants is advocated on the ground that, by preventing the anemia from which most of them would otherwise suffer, the level of health of the infant population would be raised.

The article is accompanied by charts showing the hemoglobin level in the blood at different ages, and the effect of iron treatment.

TWO CASES OF SUPRAPITUITARY TUMOUR. W. G. WYLLIE, Proc. Roy. Soc. Med. **22**:391 (Feb.) 1929.

Cases of suprapituitary tumor were reported in two girls, aged 10 and 21 years, respectively.

UNILATERAL HYPERTELORISM. V. P. H. SHELDON, Proc. Roy. Soc. Med. **22**: 393 (Feb.) 1929.

A case of unilateral hypertelorism was reported in a girl, aged 8 years who was the youngest of five children, all of normal appearance.

CYSTIC HYGROMA IN A MONGOL. R. S. CORBETT, Proc. Roy. Soc. Med. **22**:394 (Feb.) 1929.

A girl, aged 11 months, appeared healthy but was of the mongolian type. Labor had been normal. At birth, a fulness below the jaw on the left side was noticed. The swelling had varied in size, according to the report of the mother. Just before the child was admitted to the hospital, there had been a marked increase in the size of the swelling.

WILLIAMSON, New Orleans.

TREATMENT OF PNEUMOCOCCIC INFECTIONS WITH BILIARY SALTS. CASTELLANOS, Arch. de méd. d. enf. **32**:141, 1929.

Castellanos calls attention to the grave consequences of pneumococcic infections in infancy and the high mortality which persists under present methods of treatment. He offers a new mode of attack, particularly in empyemas following pneumonia, that has been developed in the pediatric department of the University of Havana. Preliminary work was based on research begun by Professor Neufeld in 1900, concerning the action of rabbit bile when added to a culture of the pneumococcus. Neufeld demonstrated that when a few drops were placed in a virulent growth, the culture was rapidly clarified and all bacteria disappeared; in addition, he proved that the solution could then be utilized in vaccinating susceptible animals. This property of bile was subsequently investigated by Nicolle and Adit-Abey, of the Pasteur Institute, who found that while the pigments had no bacteriolytic action, certain bile salts possessed this power to a marked degree. They reported the quinocholate of soda as the most active, the

taurocholate second and the glycocholate the weakest; the hypocholate had no lytic action. Later, Trucke, Cottoni and Rafael advanced the knowledge of this phenomenon. They determined that the solvent action of bile and the salts of bile depended on the virulence of the specific strain of the pneumococcus under observation; with exalted virulence produced by frequent passage in susceptible animals, the bacteria showed extreme solubility, while the saprophytic form of the pneumococcus remained insoluble.

The question then presented itself: Can this phenomenon of Neufeld be of practical use in the treatment for pneumococcic infections? The first attempt in this direction was made in 1925 by Ribadeau-Dumas and Tisseraud, who employed an aseptic bile spray in cases of pneumococcic rhinopharyngitis. Their results were on the whole favorable, but as practically all of these cases are polymicrobian, no specific action could be expected. Direct introduction of the bile salts into the blood stream would undoubtedly be extremely dangerous, on account of their known hemolytic action. Animal experimentation has shown not only laking of the blood but profound alterations in the parasympathetic system. The pulse is diminished in volume; bradycardia develops; there is necrosis of the hepatic cells, the cells of the tubuli contorti of the kidney and those of the pancreas, and coagulation of the myoplasm is common. When this element of toxicity was studied more intensively, it was found that no exact technic had been developed to measure the biliary content of the blood. The method commonly employed was based on the diminution of surface tension by the use of the stalagmomometer; other tests were founded on the well known Pettenkofer reaction. These were of value only in those cases in which the concentration of biliary salts in the blood exceeded 100 mg. per liter. In 1926, Szilard perfected a much more sensitive colorometric technic; soon afterward, the American investigators, Shattuck, Killian and Katayama, established with Szilard's method normal standards of bile concentration. They estimated that in healthy subjects there existed from 7 to 8 mg. of bile salts per hundred cubic centimeters. In cases of catarrhal icterus, these figures may increase to 2 Gm. per liter.

It is evident, therefore, that relatively enormous amounts may exist in curable conditions without the toxic manifestations described in experimental animals. The reason, of course, is that homologous bile is present in the one case and heterologous in the other. In the rabbit, for example, one may inject intravenously as much as 35 cc. of rabbit bile before fatal consequences ensue, while only 6 cc. of beef bile is needed to produce death. Castellanos undertook to treat patients for pneumococcic empyema with intravenous injections of taurocholate of soda. He abandoned this method, however, for the more direct one of injections into the area of pleural effusion, where the great diffusibility of the salt permits its disinfecting properties to be promptly demonstrated. In those cases, however, in which multiple adhesions exist and the exudate is pocketed, it is best to perform immediate pleurotomy and convert the numerous smaller collections into one sac. The dose of taurocholate employed was 5 cg. for each kilogram of body weight; in extensive accumulations, this amount may be increased to 6 or 7 cg., diluted with 20 cc. of physiologic solution of sodium chloride. Four or five injections are given daily. The author reports at length a severe case of empyema following bronchopneumonia in an infant aged 14 months, treated by intrapleural injections of sodium taurocholate, resulting in recovery.

FAMILIAL OSSEOUS DYSTROPHY. MORQUIO, Arch. de méd. d. enf. **33**:129, 1929.

Four of five children in one family developed generalized osseous dystrophy of congenital origin and identical in evolution. Search of the literature available at Montevideo failed to reveal records of similar cases. All the children were apparently normal at birth and continued in good health until the end of the first year, when notable deformities began to develop in the thorax and limbs. These manifestations continued, without any subjective symptoms, for several months and suddenly ceased. The striking feature on first examination was the aston-

ishing deformity of the neck, the arms and the legs, associated with marked disproportion in the skeletal growth; mental development was quite normal in each instance and the facial expression alert and pleasing. The thorax was box-shaped and shortened, and the head rested directly on the chest; the spinal column presented a lateral curvature as far as the lumbar region, where a moderate kyphosis appeared. The knee joints were large, particularly the internal condyles; the tibiae were not affected, but the thighs were deformed in accentuated genu valgum, which threw the feet from 10 to 14 cm. in walking: locomotion was duck-like. The articulations of the shoulder were large; motion was limited and was accompanied by loud crackling. There was total absence of the carpal bones and corresponding weakness of the musculature in the hands and forearms. The calcium content of the blood was greatly diminished, and the basal metabolic rate increased. Roentgenograms of these patients revealed clearly the evolutive march of the osteogenetic defect from the earliest stage to the point of arrest. Alterations from the normal consisted in rarefactions, deformities, bony destruction, retardation in growth and absence of essential elements, affecting especially the epiphyses of all the bones, both large and small, of the limbs. The lesions were invariably symmetrical and the process slow. The sexual apparatus in each case was entirely normal. The fifth child, while free from deformities, was a mental defective. Morquio was able to exclude all diseases of an infectious or toxic nature as the source of this peculiar invasion. He believes that the etiology lies in consanguinity, the parents being first cousins and the offspring of first cousins; he admits the possibility, however, of pluriglandular dysfunction.

AMESSE, Denver.

THE ACUTE ABDOMEN IN EARLY LIFE FROM THE PHYSICIAN'S STANDPOINT. JAMES BURNET, The Antiseptic, Madras **26**:388 (April) 1929.

The cardinal symptoms of an acute condition in the abdomen are pain, vomiting, constipation and tenderness, but any of the following conditions may give rise to these clinical features: intra-abdominal conditions: (1) appendicitis, (2) tuberculous condition of the abdomen, (3) intussusception, (4) peritonitis — pneumococcal, streptococcal, tuberculous and gonococcal, (5) intestinal obstruction, (6) ulcer, (7) congenital dilatation of the colon, (8) pyelitis and (9) worms; extra-abdominal conditions: (1) pneumonia, (2) empyema, (3) diseases of the throat, (4) Henoch's purpura, (5) tuberculosis of the spine and (6) acidosis.

SANFORD, Chicago.

CONGENITAL CYANOSIS (MORBUS CERULEUS). D. MARGARITA PELLICE ARMENGOL, Med. de los niños **30**:80 (March) 1929.

A boy, aged 12 years, presented cyanosis of the face and extremities which became intensified by muscular effort. His fingers had the drumstick, bird of prey appearance. Other symptoms were: compensatory scoliosis with concavity to the left, pain on pressure over the first three dorsal vertebrae, a soft blowing murmur over the origin of the pulmonary artery, cardiac fremitus, hypertrophy and an incease in the erythrocytes to 7,300,000. His mother died of tuberculosis, and a sister had the same disease. The author believes that this infection was an aggravating influence in the development of congenital cyanosis.

Winkel's disease and pernicious icterus with hemoglobinuria must be differentiated from this condition. Developmental defect of the interventricular septum is a frequent cause of congenital cyanosis. Serres observed that such defects rarely exist alone, stenosis of the pulmonary artery frequently being associated. Defects in the auricular septum cannot explain this condition because the defect is frequently found at autopsy. Among other anomalies which have been studied by Feer is a transposition of the great vessels. The aorta may proceed from the right ventricle and the pulmonary artery from the left. In such a case, if a

wall separates the right and left ventricle, life is impossible; but if a communication exists which allows arterial and venous blood to mix, there is a possibility of short life.

Although pulmonary stenosis is one of the most common causes of congenital cyanosis, stenosis of the aorta is rare. Hypertrophy of the ventricle is dependent on the narrowing of this orifice, combined frequently with an interventricular opening.

These cases also show pulmonary disturbances caused by passive congestion such as dyspnea, poor nutrition, a certain degree of infantilism, decrease of temperature in the extremities and, finally, an increase in the erythrocytes up to 8,000,000 as a result of faulty oxygenation of the blood.

DWYER, Kansas City.

PERIRENAL ABSCESS IN INFANCY. J. GARCIA DEL DIESTRO, Arch. españ. de pediat. **12**:784 (Dec.) 1928.

The clinical development of perinephritic abscess takes place in three stages.

In the first stage, the general symptoms of an infection predominate. This period may last for two or three weeks.

In the second stage, the local symptoms become more prominent. They consist generally of pain aggravated by movement or by pressure. The vertebral column is fixed and may show partial scoliosis. There may be contraction of the psoas muscle.

In the third stage, the patient develops unmistakable signs of renal disease. There is general bloating, swelling of the extremities and generalized edema. The prognosis is good if the disorder is discovered in time.

SCHLUTZ, Minneapolis.

URINARY DISTURBANCES IN AN INFANT CAUSED BY HEXAMETHYLENAMINE. A. BARBOZA, Arch. de Pediat. (Rio de Janeiro) **2**:244 (April) 1929.

A child, aged 11 months, had an elevation in temperature and symptoms of coryza. As the urine showed some pus cells, hexamethylenamine was given. This was followed by a suppression of urine and a temperature of 39 C. (102.2 F.). The urine that was finally passed was greatly concentrated and contained blood. All symptoms disappeared on withdrawal of the hexamethylenamine.

SANFORD, Chicago.

TREATMENT OF SERUM DISEASE. H. TATERKA and L. HIRSCH, Med. Klin. **25**: 1624, 1928.

Serum disease is more often seen nowadays because serum is used more frequently than before. The authors compared the known methods for the treatment of serum disease. The administration of calcium as a prophylactic measure did not show any remarkable results. Atropine was useless. Epinephrine had many undesirable effects, and therefore its use was rather limited. The authors found that epinephrine can be well supplanted by ephedrine. The effect of this drug is somewhat slower than that of epinephrine, but lasts longer; with repeated doses, the results are quite satisfactory.

As a preventive measure, it seems to work in some instances but fails in many others. Of course it is rather difficult to judge these results, as it cannot be foretold whether serum disease is going to develop or not. The dose for older children is 50 mg., for smaller children 25 mg. every two hours if given by mouth, and every four hours if given subcutaneously.

ZENTAY, St. Louis.

VULVITIS APHTHOSA. EMIL FLUSSER, Monatschr. f. Kinderh. **43**:123 (April) 1929.

Vulvitis aphthosa is an infection of the vulva of the same type as stomatitis aphthosa. The writer says that in the past no one realized the clinical identity

of the two conditions. He reports a cleancut case in a little girl with aphthous stomatitis who apparently spread the infection from her mouth to the genitalia. Although there is some evidence in favor of the idea that the spread is metastatic, he is convinced that in this case it was by direct inoculation.

The treatment of aphthous vulvitis is, first and foremost, to leave the patient alone. Direct applications do more harm than good. The course is from five to eight days. The course can be definitely shortened by intracutaneous injections of milk, a nonspecific milk preparation and citrated blood. Apparently the nature of the substance injected is of little importance, even distilled water having an effect. The essential feature is that a wheal must be formed which is big enough to cause a complete anemia of the underlying skin, i. e., from 0.3 to 0.5 cm. in diameter. In occasional cases, injections can be repeated in two days.

In the severest cases, one gives neoarsphenamine, but never more than 0.5 cg. per kilogram of body weight; this may be repeated in two days.

GERSTLEY, Chicago.

SORBITE AS A SOURCE OF CARBOHYDRATE IN DIABETES. S. J. THUNNHAUSER and KURT H. MEYER, München. med. Wchnschr. **76**:356 (March 1) 1929.

Storage of glycogen is the keynote of the metabolism of diabetes. Insulin is necessary for the synthesis, but not all monosaccharids build up glycogen equally. According to the authors' investigations, levulose is of greater advantage than dextrose. They now believe that they have found a sugar of great value commercially that is manufactured by I. G. Farben. This sugar sorbite is the alcohol produced by the reduction of dextrose. Cane sugar and dextrose are from four to five times as sweet as this substance, which contains 290 calories in 100 Gm. and has a pleasant taste. The authors give the results in four cases of diabetes, and the observations on the storage of glycogen in the livers of four dogs. They advise giving first from 30 to 40 Gm., then 60 Gm. a day to adults. They believe that it will become an important factor in the diet of diabetic patients.

LOCAL FAT DYSTROPHY AFTER INJECTIONS OF INSULIN. R. PRIESEL and R. WAGNER, Ztschr. f. Kinderh. **46**:453 (Oct. 31) 1928.

It appears more probable that the causative agent for local fat dystrophy is to be found in the tricresol supplement rather than in a lypolytic ferment assumed to be present in the insulin. These investigators recommend that another and less dangerous means of preservation be considered, that the same site of injection should not be used all the time and that double concentration, at least of insulin, should always be given.

A CASE OF GERODERMA GENITODYSTROPHICUM IN CHILDREN. I. ROSENSTERN, Ztschr. f. Kinderh. **46**:481 (Oct. 31) 1928.

A case of geroderma genitodystrophicum is described in a boy, aged 8 years, showing in addition to the cardinal symptoms, a senile alteration of the entire skin and abnormally small and hard testicles, a senile physiognomy and flabby musculature, with ptosis of the abdomen and a considerable diminution of strength in general, kyphoscoliosis, conspicuous deposition of fat on the hips and upper legs and an increased tolerance for carbohydrate. The signs bespoke a primary genital hypoplasia. Histologically, the skin evinced a great diminution of elastic tissue.

EXUDATIVE DIATHESIS AND BLOOD PRESSURE. A. SOKOLOW and A. ROSENTHAL, Ztschr. f. Kinderh. **46**:554 (Oct. 31) 1928.

One of the authors previously reported the results of the observation of dermographism in very young children with different forms of exudative diathesis. The dermographic reaction was obtained in these children with difficulty and then only weakly. Moreover, in a number of them, by the use of the usual and regular

pressure of moderate strength, either no skin reaction or only white dermographism was shown. With increasing age the tendency was greater for the vessels of the skin to react to a mechanical stimulation, not with dilatation but with spasm, so that the frequency of white dermographisms in such children who were more than 2 years of age reached about 88 per cent. It seemed possible to seek the cause in the contractility of the walls of the vessels of the skin (and also the smooth muscle) as a result in change of milieu, in which the dermographic reaction is concerned. Although the significance of milieu was to be looked on as unquestionable for the character of the vessel reaction, nevertheless it was necessary to find some objective evidence that the increased tendency to vessel spasm really existed in more or less extensive regions of the body. The authors therefore employed determinations of blood pressure which depends, among other things, on the vessel tone. After careful measurements of ninety-six normal infants and of eighty-nine with exudative diathesis, from the age of 1 month to 2 years, the conclusion was reached that in exudative diathesis a more or less outspoken hypertonia exists. The comparison of the figures for blood pressure and the character of the dermographic reaction showed that the highest blood pressures always were observed in the cases in which white dermographism was especially marked. In case the observations presented here are confirmed by the testing of a greater number of children, one could speak of a kind of constitutional hypertonia in exudative diathesis. Naturally, the question will arise as to the eventual interdependence of exudative diathesis and blood pressure in the hypertonia of adults.

EMERSON, Boston.

THYROXIN IN INFANTILE MYXEDEMA. J. GELDRICH, Orvosi hetil. **72**:946, 1928.

Thyroxin was given in doses of 0.2 mg. twice a day to a myxedematous child, aged 14½ years, and to a normal child, aged 13½ years. After nine days of medication the basal metabolic rate rose 60 per cent in the myxedematous child, while in the normal child it rose 14 per cent. After the medication was stopped, the basal metabolic rate returned to normal within two weeks in the normal child, while the myxedematous child had a higher metabolic rate even after six weeks. The pulse rate changed in harmony with the basal metabolism. The weight remained stationary in the myxedematous child, while it increased slightly in the normal child. The excretion of nitrogen and water increased in the first few days; later, it gradually decreased.

ZENTAY, St. Louis.

Nervous Diseases

SEX, AGE AND SEASONAL DISTRIBUTION OF TETANY IN THE ORPHANAGES IN PEKING. SHIH-HAO LIU and FU-TANG CHU, Am. J. M. Sc. **177**:559 (April) 1929.

In metabolic studies of the type of tetany that occurs in the orphanages in Peiping, Liu and Chu demonstrated that the condition cannot be accounted for solely by a low calcium intake in the diet and that a deficiency in vitamin D, as contained in cod liver oil, or lack of available ultraviolet rays in the sunlight plays a greater part in bringing about a condition of neuromuscular hyperirritability. The age of their subjects varied from 4 to 23 years. No case of tetany was found in boys before the age of 14 years. From then on, the incidence of tetany increased until it reached the maximum at 18 years. The incidence of tetany in females was distinctly higher than in males, showing that it is more prevalent among girls than boys. Tetany affects the male from 14 to 21 years of age and the female from 10 to 20 years. The incidence is much higher in winter than in summer. The examination was conducted in February and October. A comparison of the two tables of the incidence of tetany according to age and sex during these two periods shows that there is a reduction in the rate of tetany during the summer months. The facial phenomenon was demonstrated in only 9.8 per cent of the boys and in

10.9 per cent of the girls, as contrasted with the corresponding figures of 22.8 and 35.0 per cent obtained in the winter; not a single case of active tetany occurred in either male or female during the summer.

HEUNKE, Omaha.

CEREBRAL CALCIFICATION EPILEPSY: ENDARTERITIS CALCIFICANS CEREBRI. H. RAWLE GEYELIN and WILDER PENFIELD, Arch. Neurol. & Psychiat. **21**: 1020 (May) 1929.

Epilepsy in a father and all four of his children was associated in each case with changes in the brain leading to calcification of a peculiar type. In the father, convulsions did not occur until he was 48 years of age, although he had always suffered from migraine. Each child presented normal mental development up to the age of 1 or 2 years; then each showed arrested mentality. The first signs of epilepsy appeared at 6 weeks, 5 months, 5 months and 2 days of age, respectively. The convulsions varied in form, including the individual extremities and face, or the whole musculature. One outstanding feature in all these attacks was the invariable accompaniment of pupillary change. In cases 1 and 4, the pupils were greatly contracted with each attack. In cases 2 and 3, the pupils were dilated at such times. Occasionally, when attacks were slight, only the pupillary spasm appeared, constituting a curious type of "petit mal" which the mother did not fail to observe, owing to her training as a nurse. In two of the children there was a partial paralysis of one extremity, which gradually improved. Occasional swellings of the lymph glands in three of the children were noted by the mother. Severe convulsive attacks were induced in all by such intercurrent disturbances as diarrhea, tonsillitis and dentition.

In the four cases in which roentgenograms were obtained, discrete patches of calcification were found scattered through the brain, situated in general near the convexity of the cerebrum. Operative excision in the only case in which the calcification was localized resulted in great improvement. Microscopic examination showed the process to be not parenchymatous but primarily vascular. The glial changes were focused around the smallest blood vessels, and the deposit of calcium had taken place about and in the walls of these small vessels. "Cerebral calcification epilepsy" would therefore describe the obvious manifestations of the disease. The actual pathologic process involved, however, is more clearly indicated by the term, "endarteritis calcificans cerebri."

SANFORD, Chicago.

THE DEVELOPMENT OF SPEECH. JOHN A. GLASSBURG, M. J. & Rec. **129**:69 (Jan. 6) 1929.

The origin of speech is unknown. It possibly began many thousands of years ago as a vocal reflex or as a sound expression of a physiologic demand. Whenever the senses are excited or the muscles are hard at work, relief is experienced in uttering sounds; the emotions also—sorrow, joy and surprise—are apt to be accompanied by reflex exclamations. The first real utterances made by the newborn child are the sounds accompanying the physical needs of hunger, pain and cold. Speech is an acquired faculty. One is born deaf, dumb and blind, but hearing sets in from the first day. The sense of hearing is the most acute of all the senses in infancy. The child learns to speak by imitation. The average age of onset of speech is between 1 and 2 years. Girls begin a few months earlier than boys. The causes and treatment for retarded speech are discussed in the paper, as is the speech of the mental defective.

WILLIAMSON, New Orleans.

IRRADIATED ERGOSTEROL IN THE TREATMENT OF TETANY. MORRIS GLEICH and SAMUEL GOODMAN, New York State J. Med. **28**:1398 (Dec. 1) 1928.

Two cases of active tetany and one case of latent tetany are reported. In all, the blood calcium and phosphorus contents were low. Ergosterol (from 2 to 10 mg.) was given intravenously early in each case; later, it was given by mouth.

An increase in blood calcium and phosphorus was shown. After treatment with ergosterol, convulsions were relieved promptly. While the intravenous use of ergosterol is not advised as a routine measure, it may be used when life is threatened.

THE RÔLE OF ARTERIAL OCCLUSION AND ANOXEMIA IN THE CAUSE OF THE CEREBRAL DIPLEGIAS. HAROLD R. MERWARTH, New York State J. Med. **29**:1 (Jan. 1) 1929.

While cerebral hemorrhage of the new-born does cause this condition, it is probably not the only etiologic factor. Frequently the blood clots found on autopsy are small, and it is difficult to understand how they could produce a bilateral injury of the cortical areas of the leg.

The author believes that considerable torsion and twisting must be produced in the cerebral arterial system as a result of molding during labor. This would cause sufficient anoxemia to damage irreparably the nerve cells and in some cases to cause rather extensive involvement. Cerebral diplegia is frequent in premature infants, because the arteries are more fragile and more easily compressed.

AIKMAN, Rochester, N. Y.

MENINGOCOCCUS MENINGITIS: TREATMENT BY COMBINED CISTERNAL AND LUMBAR MEDICATION. M. J. FOX, Wisconsin M. J. **27**:548 (Dec.) 1928.

Because of frequent subarachnoid block in meningitis and the disappointing results after the use of lumbar medication in late cases of meningitis, a combination of the cisternal and lumbar routes is advised.

More than 400 cisternal punctures caused no ill effects. In sixty-nine cases in which this method was used, the mortality rate was 17.4 per cent.

The total dosage of serum averaged 190 cc. per patient; 33.23 per cent of the serum was given intercisternally, 23.64 per cent by the intralumbar route and the rest intramuscularly and intravenously.

JANNEY, Milwaukee.

A CONTRIBUTION TO THE CLINICAL STUDY OF SCHILDER'S ENCEPHALITIS. C. P. SYMONDS, Brain **51**:24 (March) 1928.

The author discusses the possibility that the disease first described by Schilder, in 1912, as "encephalitis periaxialis diffusa" may have a familial incidence. He describes a case proved by postmortem examination to be Schilder's encephalitis which gave a clinical picture of a progressive double hemiplegia without evidence of increased intracranial pressure. "The advanced bilateral pyramidal signs, without involvement of brain stem nuclei, suggested symmetrical disease in both hemispheres rather than a single central lesion." The diagnosis of Schilder's encephalitis was made on this basis. The family history was interesting in that, of the twelve other children, "one died in infancy from unknown causes; one died in adult life from pulmonary tuberculosis; three are alive and well; five died in infancy or childhood from causes noted as 'meningitis' or 'convulsions' without further evidence of the clinical or pathological state. The other two, a girl aged 20 and a boy aged 11, present symptoms of cerebral disease." The author suggests that their symptoms are compatible with the clinical diagnosis of Schilder's encephalitis. The postmortem examination is reported, and four photographs of sections and an ample bibliography are included in the article.

THE NARCOLEPSIES. S. A. KINNEAR WILSON, Brain **51**:63 (March) 1928.

Narcolepsies are discussed at great length: the nosology, terminology, personal illustrative cases, classic and clinical varieties, clinical symptoms (sleep, attacks of loss of tone and power, transitions and substitutions), relation of narcolepsy to other clinical syndromes (myoplegia, catalepsy, epilepsy, Oppenheim's Lach-

schlag), etiology, pathology, sleep centers and their localizations and pathogenesis. Wilson states that "with unfamiliar conditions like the 'narcolepsy,' which some persist in calling a 'disease,' attempts at identification and at nomenclature should come last, not first." The various narcoleptic syndromes offer many analogies to those usually termed epileptic. Differing in outward manifestations they still suggest that a similar function lurks behind each, though it is impossible to determine just what this is. On analysis, narcoleptic phenomena exhibit close affinity to those of epilepsy and catalepsy. The author thinks that the term "sleep" is too vaguely and inaccurately used as regards many of the symptoms classed as narcoleptic. He suggests that greater significance from the point of view of etiology and pathogenesis "attaches to the toneless, powerless, motionless awareness of numerous cases (both in 'sleep' attacks and in cataleptic attacks), than to actual sleep." An extensive bibliography completes this careful study of the narcolepsies.

ANKLE-CLONUS: THE DISTINCTION OF THE ORGANIC AND FUNCTIONAL VARIETIES. RICHARD S. LYMAN, Brain **51**:168 (June) 1928.

The ankle clonus test is important as an aid in checking up those hysterical subjects who may be malingerers or those whose clonus is due to a physiologicotoxic cause, as only patients with definite pyramidal lesions are likely to react according to the law of antagonistic reflexes. The author gives a historical review with an extensive bibliography. Ankle clonus and flexion reflex are discussed with his procedure for testing, which he claims is rapid and simple and a clinical aid in distinguishing organic from functional types of clonus. This consists of "forcible plantar flexion of the homolateral hallux while ankle-clonus is being maintained. If the clonus is then checked, an organic lesion in the central nervous system is postulated. If it persists in spite of prolonged toe-flexion, it is probably functional in origin." He further states that "the mechanism of cessation of ankle-clonus by flexion of the great toe in organic disease depends on an antagonism of reflexes. Clonus requires increased tone of the extensor muscles of the foot. Forceful plantar flexion of the hallux gives rise to the withdrawal or flexion reflex in which there is strong tonic contraction of the dorsiflexors of the foot. That flexor contraction causes reciprocal relaxation of the opposing extensor muscles with consequent disappearance of the clonus. Plantar flexion of the great toe provides stimuli which are largely proprioceptive and which make up the sensory portion of the arc relating to the flexion reflex."

OCULOGYRIC CRISES IN CHRONIC EPIDEMIC ENCEPHALITIS. P. K. MCCOWAN and L. C. COOK, Brain **51**:285 (Oct.) 1928.

The authors discuss the tonic eye spasms, "crises oculogyres," a comparatively recent phenomenon in chronic epidemic encephalitis. The paper is based on a series of 23 cases taken from a total of 136 cases of encephalitis. In all the cases, parkinsonism preceded the oculogyric crisis by periods ranging from a month to six years. These crises are in the nature of tonic seizures, which are not necessarily confined to the eye muscles, a more or less coordinated spread to the muscles of the head and neck not being uncommon. Psychic distress of various kinds is common. The fixation of the eyes, e. g., forced convergence, frontward stare, etc., is important in determining the site of the lesions. Length of attack varies from a few seconds to several hours. The causal lesion of oculogyric crises lies in an associational mechanism situated above the four supranuclear centers subserving conjugate movements, the theory being accepted that there is a supranuclear center for lateral conjugate movements and one for vertical movements on each side. In a certain number of cases, crises tend to disappear with time and if scopolamine hydrobromide is administered hypodermically under careful supervision in selected cases. Photographs, case reports, and a bibliography are included in the article.

LUCAS, San Francisco.

PSYCHOANALYSIS APPLIED TO ANOREXIA IN CHILDREN. A. BRETSCHNEIDER, Arch. f. Kinderh. **86**:94 (Jan. 25) 1929.

Bretschneider believes that anorexia in children is sometimes a symptom of a mental unrest which may be discovered by psychoanalysis. He gives two cases in which an interpretation of the child's dreams disclosed the cause of the trouble.

INFLUENZAL MENINGITIS IN INFANTS. E. NEDELMANN, Arch. f. Kinderh. **86**:98 (Jan. 25) 1929.

Nedelmann insists on a strict distinction between influenzal and suppurating meningitis. He reports eight cases. In six, the blood test was positive. The author believes that influenzal meningitis is a septic disease in which the inflammation of the meninges, due to metastasis, becomes preponderant and causes death. The blood culture always contained Pfeiffer's bacilli as soon as the bacteriologic examination indicated an infection by this cause. None of the strains of this organism proved to be hemolytic.

DEGENERATION AND DEFORMITIES OF THE FOOT. A. FISCHER, Arch. f. Kinderh. **86**:109 (Jan. 25) 1929.

Fischer presents three cases of various deformities of the foot. In the first case the child was a typical mongolian idiot, and in the last two, the children were definitely subnormal. He states his belief that deformities of the foot are common in degeneracy; to support this view, he quotes the observations of Féré and Demantké of 35.3 per cent of flatfoot in epilepsy, and of van der Scheer of 2.3 per cent of deformity of the foot in 300 mongolian idiots.

THE INFLUENCE OF HOME AND INSTITUTIONAL TRAINING ON THE CHILD'S CHARACTER. F. HAMBURGER, Arch. f. Kinderh. **86**:161 (Feb. 22) 1929.

The most important years from the standpoint of character building are the first six. It is therefore in these years that the most effort must be made to shape the character of the child. Institutional training is not satisfactory in this respect until after six years, when correct habits are well taught. The earlier years are better controlled by the mother in the family circle.

SANFORD, Chicago.

PSYCHOLOGIC INVESTIGATIONS ON THE EFFECT OF SEA CLIMATE. HEINRICH VOIGTS, Monatschr. f. Kinderh. **43**:97 (April) 1929.

This study is a continuation of previous observations made by Voigts on the effect of a change of environment from the interior of the country to the seashore. The study is concerned with groups of children sent from various parts of Germany to the bathing resort, Travemunde, which is near the city of Lubeck. Previous studies were concerned more with the physiologic, these studies more with the psychologic effects. The first tests used were those devised by Kraepelin and employed in the German schools; these were apparently like the Binet-Simon tests. There was no question that the children at the seashore showed reactions decidedly superior to those which they showed in their native environment. This held true even in the groups from Lubeck, though it is only a short distance from the ocean.

Of course at the seashore the children were outdoors all day. As Voigts says, if one goes to the seashore and sits in his room all day, one can expect no results. Unquestionably, though, the sea air itself has a definite effect. The quality does not penetrate far inland; furthermore, due to reflection from ocean and sands, the sea air is richer in ultraviolet rays.

The psychologic studies consisted in asking the children to write short paragraphs on such subjects as "Bathing," "Our Sailing Trip" and "My Experiences on the Beach." All their answers were characterized by a predominant realism. Numerous examples of their replies are given, including some written during each season of the year.

Concerning the behavior of the children, July seemed to be the worst month. In both the late spring and the early autumn the children were quiet and obedient and seemed to be soothed and rested. In July, due perhaps to the crowds at the height of the bathing season or to the heat, the children were restless, irritable and less obedient.

The writer believes that the child's disposition is powerfully affected by the beauty of nature and that the best results were obtained in early autumn, which is the most beautiful season in that particular resort.

THE EAR AS AN EROTIC ORGAN IN CHILDREN. JOHANN SUSSMANN GALANT, Monatschr. f. Kinderh. **43**:130 (April) 1929.

Sexologists have described various erotic zones in the human body, but apparently have overlooked the ear. Anthropologists and criminologists have studied the ear from the point of view of atavistic peculiarities, and psychologists have looked at the ear for stigmas of degeneration. Galant now stakes out his claim for the sexologists.

His first arguments are based on Russian poetry. He quotes four lines from the poet Igor Sewerjanin:

> "I gripped little Peter
> By his ear
> Then I whispered into it,
> 'I'm going to bite you.'"

Further evidence Galant finds in the tendencies of all people to decorate their ears. Third, he quotes an immortal case of a little Russian girl who played about her naked father while he was on the bathing beach, continually striking his penis and then asking him to kiss her ear. Galant believes that the ear as a sexual organ has been long overlooked. By lending itself to decoration, kisses, biting and sucking, the ear is an erotic organ par excellence. GERSTLEY, Chicago.

THE CLINICAL VALUE OF SENSITIVITY TO PRESSURE OF THE ACHILLES TENDON AND OF THE TESTICLE. THOMAS C. HUNT, Wien. klin. Wchnschr. **42**:299 (March 7) 1929.

Abadie's sign, diminished sense of pain when the Achilles tendon is compressed, which is supposed to indicate the presence of tabes, was found in 35 per cent of 100 patients who had neither syphilis nor organic nervous disease. Pitre's sign, diminished sense of pain when the testicle is compressed, was found in 18 per cent of 50 men who had neither syphilis nor organic nervous disease.

CLAUSEN, Rochester, N. Y.

INTRACEREBRAL CALCIFICATIONS IN CHILDHOOD. W. SAUER, Ztschr. f. Kinderh. **46**:457 (Oct. 31) 1928.

A short review is given of the occurrence of intracerebral calcification. Of the two cases here reported, the first showed a calcified multiple cerebral tubercle, the other a calcification or ossification, the etiologic cause of which could not be found. The diagnosis was not confirmed by postmortem examination.

EMERSON, Boston.

STIMULANT DRUGS IN PARALYSIS OF THE RESPIRATORY CENTERS. G. JÁNOSAY, Orvosi hetil. **73**:43 (Jan. 12) 1929.

In several cases of failure of the respiratory center, intralumbar and intracisternal injections of alpha-lobeline and caffeine were made consecutively. The reactions on the part of the respiratory center were carefully registered. Three milligrams of alpha-lobeline given in the cisterna produced a marked excitation of the respiratory center, while 5 mg. given by the intralumbar route did not show any results even after an hour. The author thinks that the best method of using stimulant drugs for the respiratory center is through the cisterna. The explanation lies in the close proximity of the medulla to the cisterna.

INTRAVENOUS INJECTIONS OF NEOARSPHENAMINE FOR CHOREA MINOR. J. BÓKAY, Orvosi hetil. **73**:465 (May 18) 1929.

Bókay decides that his former method of treatment for chorea is as reliable as any of the new ones recommended, particularly for severe forms of the condition. He has not seen any unfavorable effects. His dosage is from 0.1 to 0.3 Gm. every five or seven days. Most of the patients improve in from three to six days. If the treatment is started at the early stage, the results are more satisfactory. The general condition of the patient is beneficially influenced by neoarsphenamine. Recurrences are not influenced by the treatment. The action of neoarsphenamine does not seem to be on the lines of changing of metabolism or of chemical reaction of the blood.

ZENTAY, St. Louis.

Diseases of the Ear, Nose and Throat

ROENTGEN OBSERVATIONS ON SAFETY PINS AS FOREIGN BODIES. WILLIS F. MANGES, Arch. Otolaryng. **9**:245 (March) 1929.

In Jackson's clinic at the Jefferson Hospital in Philadelphia, about 12 per cent of the foreign bodies found in the gastro-intestinal and respiratory tracts are safety pins.

The roentgenologist's function in cases of foreign bodies is to determine the presence or absence of such a body, its location, relative position, size, condition, local tissue reaction, and variation of position and progress from day to day. He helps during the removal of the object with the fluoroscope, detailing by means of a two plane screen the progress of removal. Determination of the exact location of the foreign body is imperative if it is below the larynx, and the distribution of the parts must be known in order to remove it skilfully and safely. Films should show shadows of the larynx and the trachea. A bismuth compound taken by mouth will often aid in localization.

Most types of foreign bodies tend to lodge in some particular part of the respiratory tract, as, for example, the peanut in the bronchus and coins at the level of the suprasternal notch. However, the safety pin may be found anywhere from the nasopharynx to the distal part of the lung or any part of the gastro-intestinal tract. The age limits reported for the patients in whom foreign bodies are found are from 6 weeks to 46 years, and pins have remained in the body from one day to thirty-six years. In the trachea the two branches of the safety pin are most often in the sagittal plane, though foreign bodies often rotate when they reach the bifurcation so that the branches of the pin are then disposed laterally.

By using a distance of 36 inches in taking films, one may obtain a fairly accurate idea of the size of the foreign body.

Complete cooperation between the roentgenologist and the bronchoscopist is imperative, and to make this possible it is best to outline their plan of attack before removing a foreign body. They must be able to understand each other completely.

CARCINOMA OF THE LARYNX IN THE YOUNG. FREDERICK A. FIGI and GORDON B. NEW, Arch. Otolaryng. **9**:386 (April) 1929.

Carcinoma of the larynx is so rare in young persons that it may be overlooked. In the ten years from 1919 to 1929, one patient, aged 15, and one, aged 24 years, were seen at the Mayo Clinic. Case reports of both are given by the authors.

Laryngeal carcinoma has to be differentiated from syphilis, tuberculosis, blastomycosis, rhinoscleroma, chronic laryngitis, papilloma, fibroma, angioma and prolapse of the ventricle. One should not hesitate to do a biopsy for diagnostic purposes if necessary. Complete laryngectomy, early and properly done, is justifiable once the diagnosis is established.

BARNETT, Chicago.

METHOD OF REMOVING FOREIGN BODIES FROM THE NOSE. W. OBADALEK, München. med. Wchnschr. **76**:634 (April 12) 1929.

The following method is proposed for those cases in which the child is obstreperous, should not have an anesthetic and has pushed a fragile foreign body, such as clay, mud or chewed-up paper, into the nose. The child lies on the bed, with his head lowered, and a gag is put in his month. The index finger of the left hand is introduced in the mouth so as to cover the lower part of the nose in which the body is located. Then a rubber catheter of the right size is inserted, with the left index finger as a guide, and the foreign body is gradually pushed out of the nose. During this time a piece of gauze must be over the mouth so that the foreign body cannot drop into it after coming out of the nose. The author has tried the method with success in thirty cases.

SCHORER, Kansas City, Mo.

CYSTS OF THE TONGUE IN CONGENITAL STRIDOR. HERMANN POST, Ztschr. f. Kinderh. **46**:566 (Oct. 31) 1928.

Since the patient in the case reported was treated by two local practitioners and then by a pediatrician before he was admitted to the clinic, the author would urge the constant and careful digital examination of the throat in cases of congenital stridor, because these patients can be cured by a simple surgical procedure which every practitioner is capable of doing.

EMERSON, Boston.

Diseases of the Skin

THE PROTEIN CONTENT OF THE BLOOD SERUM OF INFANTS, PARTICULARLY OF ECZEMATOUS INFANTS. ERNA LEVY, Rev. franç. de pediat. **4**:576 (Oct.) 1928.

In nine cases of infantile eczema and in thirty other cases, serum protein was determined by the micro-Kjeldahl method. It was found that in general the quantity of protein in the serum increased gradually from 4 or 5 Gm. per hundred cubic centimeters during the first month of life to 7 or 8 Gm. at the twelfth month, this being the normal figure for the adult. With one exception, the cases of eczema showed a relatively low protein content regardless of the ages. Those in which the condition was of long duration showed the lowest figures. This hypoproteinemia is probably only one of the manifestations of eczema and is not to be considered as a basic cause.

HERPES ZOSTER AND VARICELLA. M. GIUFFRE, Rev. franç. de pediat. **4**:623 (Oct.) 1928.

After a review of the literature on the relationship between varicella and herpes zoster, none of which is convincing, the author reports two incidents which came under his own observation. A boy, aged 3 years, presented a typical picture of herpes zoster; ten days later, his younger brother developed unmistakable varicella. The children had not been with other children, nor had there been

another case of varicella in the neighborhood. In another family a child, 2 years of age, developed varicella. Inquiry revealed that fifteen days before, his father had had herpes zoster. The author suggests that there may be a form of varicella that is indistinguishable from herpes zoster.

LESLIE, Evanston.

DERMATOMYOSITIS. H. LEHMKUHL, Arch. f. Kinderh. **86**:179 (Feb. 22) 1929.

Lehmkuhl's patient suffered from degeneration of function in all muscle groups, accompanied by considerable pain. The skin was red and rough. There was no elevation of temperature. The blood count showed 4,000 white cells, 4,000,000 erythrocytes and 60 per cent hemoglobin. A differential count revealed 60 per cent leukocytes, 32 per cent lymphocytes and 3 per cent eosinophils. The Pirquet reaction was negative. The urine, stools and spinal fluid were normal. The differential diagnosis lay between trichinosis, which was ruled out by the blood picture, a tumor of the spinal cord or encephalitis, which were ruled out by the spinal puncture, and postencephalitis, which was ruled out by the temperature.

SANFORD, Chicago.

HEREDITARY PEMPHIGUS. KARL HEINZ KUSE, Inaug. Dis., Leipzig, 1929.

Kuse reports a case of pemphigus in a male infant which developed shortly after birth and was still present at the ninth month. The mother had noted a similar condition in a sister who had died of bronchopneumonia at 4 days of age. The distribution was general. Bacteriologic investigation of the content of the bullae revealed nothing. The Wassermann reaction was negative.

The picture fits in with that described by Mautner, in 1922, as hereditary pemphigus. In contradistinction to epidermolysis, there was no atrophy of the skin, scarring or pigmentation, nor did the blisters appear following mechanical stimulation. No satisfactory treatment is as yet known, and the prognosis is unfavorable.

LESLIE, Evanston, Ill.

Surgery and Orthopedics

NON-OPERATIVE TREATMENT OF INTUSSUSCEPTION. GEORGE M. RETAN, New York State J. Med. **28**:1408 (Dec. 1) 1928.

In two cases, intussusception was reduced by the injection of a solution of barium sulphate in water into the rectum. The child is placed on a horizontal fluoroscopic table, and the column of barium sulphate is observed while manual pressure is applied to force the fluid upward. If the obstruction is relieved, the colon should be filled with barium sulphate and carefully studied. When the colon cannot be completely outlined, operation is advised. In the third case reported, manual pressure failed to reduce the obstruction; the colon could not be completely visualized. Operation demonstrated that the ascending colon and the first part of the transverse colon were twisted into a mass and were blue. The twisting was relieved and the patient recovered. The author states his belief that constant observation under the fluoroscope makes the method safe.

AIKMAN, Rochester, N. Y.

Miscellaneous

ROENTGEN DIAGNOSIS OF FOREIGN BODIES IN THE ESOPHAGUS. WILLIS F. MANGES, Am. J. Roentgenol. **17**:44 (Jan.) 1927.

Foreign bodies lodge in the esophagus because (1) they are too large to pass, (2) they become more or less embedded in the mucous membrane, (3) the lumen is narrowed by some other lesion or (4) they may perforate and project into the periesophageal tissues. The most common foreign body of childhood is the large one causing partial or complete obstruction of the esophagus, and the most

common site of lodgment is at or a little above the level of the suprasternal notch. Both anteroposterior and lateral views should be taken, and the entire gastrointestinal tract should be included in the examination before a foreign body is ruled out. The swallowing of bismuth mixture is valuable in the final localization and diagnosis. Capsules of bismuth must be used cautiously in the presence of such bodies as bits of glass because of possible increase of trauma. Complications are: (1) acute hunger and thirst; (2) aspiration of particles of food into the trachea and bronchi, as a result of esophageal regurgitation, causing bronchial or pulmonary infection; (3) injury to the esophageal wall and secondary stricture due to a foreign body or unwise attempts at its removal; (4) periesophageal infection including pleuritis, and (5) infection following perforation and usually requiring emergency surgical treatment. The history and symptoms are valuable aids in diagnosis.

ROENTGEN RAY TREATMENT OF LESIONS OF THE LYMPHOID TISSUE. W. A. EVANS and T. LEUCUTIA, Am. J. Roentgenol. **17**:54 (Jan.) 1927.

Experiment has shown that there are: (1) extreme sensitivity to the irradiation of both the circulating and the lymphocytic elements of the lymphoid center with rapid destruction after too much irradiation, (2) great recuperative and regenerative power of the lymphoid tissues and (3) definite stimulation by small doses of roentgen rays. For this reason, treatment resolves itself into destructive and stimulative irradiation, the latter involving the protective action or mechanism of local tissues. The immunizing process consists of the antibody production due to substances liberated from the destroyed cells and the lymphocyte stimulation by different biologic, chemical and physical agents. Lymphoid lesions are classified as (1) inflammatory, which include infectious lymphadenitis, both granulomatous and hyperplastic tuberculous lymphadenitis, (2) neoplastic, which include Hodgkin's sarcoma, large round cell sarcoma, lymphosarcoma and small cell sarcoma and (3) conditions occupying an intermediate position such as lymphatic leukemia, pseudoleukemia, Hodgkin's disease, etc. The treatment outlined for the various forms varies from the minimal doses in infectious lymphadenitis to massive doses in early and mediastinal Hodgkin's disease. Roentgen therapy is of no value in acute lymphatic leukemia. An extensive bibliography is included.

STULIK, Chicago.

DRYING UP THE BREASTS. WALTER METCTZ, München. med. Wchnschr. **76**:782 (May 10) 1929.

For a number of reasons, drying up the breasts is necessary. There may be some objection to dieting and limiting of the fluid intake, although the breasts are painful. Camphor is supposed to decrease the production of milk. Diuretics have been used successfully. A 10 per cent solution of sodium mercurisalicyllallylamido acetate injected intramuscularly or intravenously is more efficient than any method that the author has tried. The urinary output is generally increased. The secretion of milk decreases within twelve hours, and in two days the desired result is accomplished. Irritation of the kidney, enteritis and stomatitis are contraindications to the use of this drug.

SCHORER, Kansas City, Mo.

STANDARDIZATION OF THYROID PREPARATIONS. A. STASIAK, Orvosi hetil. **72**:948, 1928.

Standardization of thyroid preparations is best done on the basis of their iodine content. Biologic methods have to be used if there is any suspicion of adulteration with organic iodide not derived from the thyroid gland. On the label of thyroid preparations the iodine content should be always given exactly.

ZENTAY, St. Louis.

THE DIAGNOSTIC VALUE OF D'ESPINE'S SIGN IN ADULTS. O. HORAK, Čas. lék. česk. **65**:953 (June 18) 1926.

The author cites various opinions as to the value of D'Espine's sign in children and adults. In adults, increased bronchophony and recognition of the spoken sound are present to the fifth dorsal spine; if they are present below this, the condition is regarded as pathologic and pathologic changes in the mediastinum or possibly aortic dilatation, or an inflammatory reaction are indicated. The author suggests the use of some monotone phrase such as tata lata to obviate mistaking intonation changes for the change from the nasal to the clear sound in the positive D'Espine reaction. Although children were not examined, the first dorsal spine for infants, the second for children up to 8 years and the fourth in older children are given as the normal limits for D'Espine's sign.

STULIK, Chicago.

THE EFFECT OF ULTRAVIOLET LIGHT ON THE BODY AND GROWTH OF THE CHILD. H. KOEPPE, Arch. f. Kinderh. **86**:81 (Jan. 25) 1929.

The twelfth article of this series is devoted to a discussion of the results reported in the former articles.

THE MORTALITY OF INFANTS IN MECKLENBURG BEFORE AND AFTER THE WAR. H. BRUNING and E. MAHLO, Arch. f. Kinderh. **86**:122 (Jan. 25) 1929.

The authors state that the mortality was less in the period after the war than in the period before the war, but that the deaths in the first month of life were higher. This conclusion was published in the *Archiv für Kinderheilkunde* (**86**:1 [Dec. 28] 1928). They now analyze these deaths in the first month of life. In the period from 1885 to 1887, on the first day of life, new-born infants showed a mortality of 4.7 per cent as compared with 10.7 per cent in the period from 1925 to 1927. On the first two days, this proportion was 8.5 per cent to 17.3 per cent. For the entire period from one week to thirty days after birth, it was 16.9 per cent to 15.5 per cent. The author calls attention to the fact that the mortality for the period from three to six days after birth in the period before the war was one in twelve, and that after the war it was one in six; in other words, it doubled during this period.

SANFORD, Chicago.

EXPERIENCES WITH IRRADIATED ERGOSTEROL. E. BAHL, Ztschr. f. Kinderh. **46**:563 (Oct. 31) 1928.

Irradiated ergosterol in the form of pastils cures craniotabes in three weeks and reduces the skeletal changes in the x-ray picture to normal in from six to eight weeks, the serum phosphate reaching normal values in from eight to fourteen days. There exists only a slight difference in the period of convalescence for severe and for mild rickets. Little progress in healing is seen at the beginning of the treatment; however, a cure is quickly effected, and though irradiated ergosterol may be used for months, harmful results are not observed. In one case of rickets associated with severe anemia the period of convalescence was extraordinarily long while in a similar case, after a blood transfusion, a sudden improvement occurred.

EMERSON, Boston.

Society Transactions

AMERICAN PEDIATRIC SOCIETY

Forty-First Annual Meeting, May 20-22, 1929

First Session, May 20, 9 a. m.

THE AMERICAN PEDIATRIC SOCIETY AND PREVENTIVE PEDIATRICS. PRESIDENTIAL ADDRESS. DR. HENRY L. K. SHAW.

This article was published in full in the July, 1929, issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

SPONTANEOUS BACILLURIA AND PYELITIS IN THE RABBIT: ITS RELATIONSHIP TO THE MODE OF INFECTION IN DISEASE OF THE URINARY TRACT. DR. H. F. HELMHOLZ.

This paper will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

DISCUSSION

DR. OSCAR M. SCHLOSS, New York: For a long time, I have been impressed by the infrequency with which pyelitis is found in the postmortem room in contrast to the frequency with which it is diagnosed during life.

During the past five years, Dr. James Wilson and I have conducted studies which have convinced us that pyelitis is an infrequent disease in young infants and children.

Examination of the kidneys of most patients who showed pyuria during life disclosed not pyelitis but a true suppurative interstitial nephritis. It is our belief that such lesions and not pyelitis are usually responsible for pyuria in young patients. We do not deny that pyelitis may occur, but we believe, to say the least, that it is exceedingly rare.

DR. JOSEPH BRENNEMANN, Chicago: Were these rabbits male or female, and did this have any bearing on the incidence of bacilluria?

DR. H. F. HELMHOLZ, Rochester, Minn.: The majority of the rabbits were female, but it did not seem to make any difference with regard to the infection.

I quite agree with Dr. Schloss that the lesions which predominate in infants are those of the kidney. In considering how the infection gets to the kidney, one must differentiate sharply between the infection with the streptococcus and the staphylococcus groups and that with the colon bacillus. For some time I have been interested in their relationship, but so far I have not been able to determine what it is.

Urinary cultures are difficult to make because of the overgrowth of the colon bacillus in almost all instances. I have had two cases in which the infection started with the staphylococcus which was gradually replaced by the colon organism. It is known that the colon bacillus is much more likely to get into the blood during the first week of life than at any other time, because of the permeability of the gastro-intestinal canal. Yet in two cases a staphylococcus was found in pyelitis of the new-born. In one, the infection was limited to the bladder for a period of two years.

I feel, as does Dr. Schloss, that pyelitis is a clinical term and that it certainly is not correct pathologically. I do not believe that there is sufficient evidence at present to warrant definite conclusion as to the mode of infection in cases in human beings.

EFFECT OF TONSILLECTOMY AND ADENOIDECTOMY ON DIPHTHERIA IMMUNITY. DR. BELÁ SCHICK and DR. ANNA TOPPER.

This paper will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

DISCUSSION

DR. L. R. DEBUYS, New Orleans: Were cultures made from the tonsils which were removed? Was a history secured with regard to any illness which the children might have had during the interval between the two periods in which they were examined?

DR. HENRY F. HELMHOLZ: I should like to ask whether in any case the Schick test was done at a shorter interval than six months.

DR. BELÁ SCHICK, New York: Nothing especially was known about illnesses occurring during the interval. The children had the usual slight infections of childhood. There was no serious sickness, pneumonia or any such condition. The interval was almost always six months. Occasionally, we had some children who came six and one-half or seven months after tonsillectomy.

THE RÔLE OF TRANSFUSION IN PEDIATRIC PRACTICE. DR. GROVER F. POWERS.

The author reviews his experience with the use of citrated blood for transfusions in sick children. The merits of the procedure in the treatment of abnormal conditions of the blood, malnutrition, surgical condition, "alimentary intoxication" and infections are discussed. The general condition of the patient rather than special signs and symptoms as the indication for transfusion is stressed. Also, the great importance of skill and care in carrying out the procedure is emphasized. Statistics are given concerning 313 transfusions performed in the pediatric service of the New Haven Hospital in 1928.

DISCUSSION

DR. CLIFFORD G. GRULEE, Chicago: My associates and I have been using transfusion a great deal for a number of years, but it has been nearly always intraperitoneal; so I do not know whether we could compare our results with these of Dr. Powers. In several respects, the results have appeared to us as being entirely different from those he mentions.

I may be wrong in my interpretation of what he means by malnourished children, but I am following the Finklestein classification of decomposition. We thought for a time that we were getting results in these children. In looking over the charts and following the cases through, we came to the conclusion that we were getting practically none at all.

This, of course, refers to intraperitoneal and not to intravenous use of blood.

Another point is with respect to the type of anemia which Dr. Powers has spoken of as nutritional. It seems to me that one will have to make a definite distinction in the secondary anemias of infancy between those which occur in the premature infant and which are nearly always of the chlorotic type, and those which are purely of a nutritional nature. The purely nutritional anemias, in my experience, have been exceedingly uncommon.

We have had no results in other secondary anemias which compared with the results we obtained in those cases which showed the chlorotic type that occurs in the older premature infant. Almost without exception, our patients with that type of anemia have shown marked improvement after intraperitoneal transfusion.

As to the cases of erythroblastosis, we have had uncertain and irregular results.

DR. KENNETH D. BLACKFAN, Boston: My views relating to transfusion are changing, because of the extraordinary, often miraculous results seen after whole blood is given into the veins. I cannot believe that the beneficial results are those purely of coincidence.

Dr. Powers mentioned the value of transfusion from the point of view of increase in the blood flow. I think that in the premature infant this is an extraordinarily essential point, when one recalls that in the early life of the premature infant there is a low plasm protein, together with other signs seen so commonly with slow blood flow and other symptoms.

In another group of cases, those showing septicemia, the use of one or two transfusions is sometimes most gratifying. It seems, therefore, that antibactericidal or antitoxic factors may be responsible for the beneficial effect.

In regard to the intraperitoneal injection of the blood, it has a specific use, as I think Dr. Grulee has emphasized. I do not think that it is effective intraperitoneally, particularly if one is looking for its beneficial results in increasing blood flow and from its antibactericidal power.

DR. FREDERICK W. SCHLUTZ, Minneapolis: It has been my experience that the direct transfusion is superior to all other procedures for quick results. I have had the same excellent results mentioned by Dr. Blackfan and Dr. Powers. This is particularly true at times in cases of septicemia.

The direct transfusion with properly matched blood of the donor is not a difficult procedure with the type of apparatus at present available, and can easily be carried out if one is reasonably skilled. It has been my impression that the direct transfusion is superior to transfusion with citrated blood and much superior to the intraperitoneal transfusion.

The transfusion with citrated blood is the method of choice if one is not sufficiently skilled in giving the direct transfusion or if a suitable apparatus for giving it is not available.

DR. ROWLAND G. FREEMAN, New York: I agree with Dr. Powers in the great value of blood serum. The younger the child, the better are the effects usually obtained. I think that the most remarkable effect is in hemorrhage in the new-born, which is usually controlled immediately by the proper use of blood serum.

Also, in these cases, blood serum or whole blood injected into the muscle will often give satisfactory results.

DR. THOMAS B. COOLEY, Detroit: My associates and I have been using transfusion for several years in a variety of cases, trying to find out when it was effective. I want to speak of one thing in addition to the things that have been spoken of, in which I agree with Dr. Blackfan: that is, in connection with bronchopneumonia in young children, in which one might hope that the transfusion of adult blood would be of great value and more resistant than the blood of the infant. But we have been sadly disappointed.

We have practically come to the conclusion that in that particular group of infectious conditions nothing important resulted from the transfusion, no effect of starting convalescence such as one might hope for, and I believe that we did not lower the mortality rate.

On the other hand, in patients with bronchial pneumonia to whom we gave a transfusion at the beginning of convalescence, after a long course of illness, we did hasten convalescence. In this group, in which we really hoped to accomplish something, the hastening of convalescence was all that we did accomplish.

DR. WALTER LESTER CARR, New York: During the past year, I have been interested in two groups of patients who have benefited by transfusion. In the first group, children with acute mastoiditis, transfusion was given before and after operation for those depleted by infection. The results have been satisfactory. Weak and anemic children with protracted pneumonia and children with a delay in healing have made a more rapid recovery after transfusion.

The second group of cases in which we have used transfusion with benefit has been a miscellaneous group, in which the condition was not classified as fatal: children with malnutrition and low grade infections. In leukemias and similar cases, I have found only transient benefit.

At one of the maternity hospitals, my associates and I are studying hemorrhagic conditions of the new-born, almost entirely mechanical, and are using blood in most of the infants injured. We are observing a series, and as we watch the infants when they return to the clinic we have found that many of these children have done better, even though subject to conditions of pressure and other factors that have to do with hemorrhage, than some of the normal children born without a record of birth trauma.

DR. A. GRAEME MITCHELL, Cincinnati: In this discussion it might be well to call attention to an additional help in transfusion which has not as yet been mentioned but which I am sure is not infrequently employed. This consists in transfusion from a donor who has been vaccinated with the micro-organisms recovered from the patient—a procedure that is applicable in certain of the more chronic infections. Somewhat analogous, too, is transfusion from a donor convalescent from the disease with which the patient is suffering.

DR. HENRY J. GERSTENBERGER, Cleveland: I should like to ask Dr. Powers whether sometimes he has had the impression that, in giving transfusion to patients with septicemia, the transfusion has been harmful.

DR. J. BUREN SIDBURY, Wilmington, N. C.: I wish to report one case of toxic typhoid fever in a child, aged 7 years. She was given two exsanguination transfusions at five day intervals. The first was given at the end of the first week, and 650 cc. of blood was given and 350 cc. withdrawn. At the second transfusion, five days later, 500 cc. was given and 250 cc. withdrawn. There was no marked change in the condition. She was delirious, picked at the bedclothes, and had a temperature ranging from 103 to 105.

Five days following the second transfusion, she had an intestinal hemorrhage which resulted in considerable shock. The following day she was given a transfusion of 500 cc. of blood from a donor who had recently (three weeks ago) taken three injections of typhoid vaccine. Forty-eight hours after this transfusion, her temperature came to normal and never went above 100. She made a rapid and complete recovery.

The amount of blood that I use for transfusions in infants is 10 cc. per pound of body weight. To some dehydrated infants I give as high as 12 or 15 cc. per pound. I believe that 10 cc. per pound is a safe rule to follow. It is possible that more than this amount may embarrass the right side of the heart. I have had this happen and have withdrawn about 50 or 100 cc. following a transfusion with immediate relief to the embarrassed circulation.

I feel that careful cross matching of bloods before each transfusion, regardless of the age of the patient, is most essential. The reaction of hemolysis as well as agglutination should be carefully looked for by the microscopic and not by the macroscopic method. Reactions following transfusions are minimized if careful matching is followed.

DR. HENRY F. HELMHOLZ: I should like to call attention to the use of acacia in intravenous therapy. It has fallen into disrepute because of the many serious reactions that followed the administration of impure solutions. For several years, my associates and I have been using a purified acacia and have had no severe reaction following its use. I believe that there is a definite field for the injection of dextrose acacia. It has appeared to us that in some cases of shock just as good results are obtained from the injection of dextrose acacia as from blood transfusion.

DR. BELÁ SCHICK: For three years, my associates and I have continued to have excellent results with blood transfusion in anemic children with unresolving pneumonia. Dr. Murray Bass, of my service, has published a report of this indication for blood transfusion.

I should like to ask Dr. Powers what his results are in such cases.

DR. JULIUS H. HESS, Chicago: I should like to ask whether Dr. Powers matched all the bloods in the young infants. My associates and I are in the habit of limiting the quantity of our individual transfusions to a maximum of one fiftieth of the body weight. Dr. Powers has used as high as one twentieth in some of his transfusions. I have seen several bad results following too large a transfusion.

DR. MCKIM MARRIOTT, St. Louis: There is another type of case in which blood transfusion is valuable; that is, in nephrosis accompanied by edema, low plasma protein and a low blood volume. By restoring plasma protein, transfusion tends to decrease edema. A further effect of transfusion is probably to increase

the resistance to infection, which is always lowered in these cases. I use transfusions regularly in nephrosis.

Dr. Grover F. Powers: In regard to what Dr. Grulee said, I can only remark that our experience with intraperitoneal transfusion is a bit discouraging, and that it is seldom used; indeed, I feel opposed to intraperitoneal transfusion except as already discussed. Cole and Montgomery emphasized the outstanding appeal which intraperitoneal transfusion makes, namely, the ease with which it may be carried out in house practice. I think that the blood should be put into the vascular system, where it properly functions.

My experience in infections has not been altogether satisfactory. I took time to show two charts, one which seemed to show a phenomenally favorable reaction to transfusion and the other, an identical reaction to intravenous injection of dextrose. I tried to emphasize in the conclusions the rôle transfusions play in the group of infections as I view the matter. I cannot answer the question conclusively from the standpoint of pneumonia. This I will repeat. In such conditions as septicemia and infections of that type, I know of no better treatment. Until the contrary can be proved, I think that transfusions should be used.

We cross-agglutinate the blood of every child to whom transfusion is to be given.

With regard to veins, the ones most frequently used are those of the elbows, ankles, wrists and the jugulars and femorals.

In regard to nephrosis, the results have not been satisfactory. An outstanding example was in a child who was tremendously edematous and who had marked diuresis following only one of many transfusions.

The Basal Insensible Perspiration of Infants. Dr. Samuel Z. Levine, Dr. James R. Wilson and Margaret Kelly.

Studies with the respiration chamber and with the calorimeter have demonstrated a striking correlation in the human subject between the production of heat and the loss of heat by way of vaporization of water through the skin and lungs. The technical difficulties inherent in the measurement of water by the chamber method led to the use of an accurate balance for determining the insensible perspiration of infants. Control experiments afforded evidence of the reliability of the method and demonstrated that with careful technic and under standard environmental conditions, infants maintain a basal rate of insensible perspiration which remains essentially constant from day to day.

Thus far, the insensible loss of eighty-one infants from 9 days to 25 months of age, in various states of nutrition and free from disease, has been determined in eighty-seven basal observations of from one to three hours' duration by the method of weighing. Within the age range studied, the basal rate of insensible loss averaged 1.1 Gm. per kilogram of body weight per hour for the infants in good or fair nutrition and 1.31 Gm. per kilogram per hour for the infants who were 35 per cent or more underweight for their age. The average figure for the normal infants is approximately double the basal figure of 0.5 Gm. per kilogram proposed for adults by Benedict and Root and more recently by Myles. The mean deviation from the average values was less than ± 9 per cent in both groups of measurements.

The basal rate, on the basis of surface area, rose steadily with increasing age, averaging 16.2 Gm. per square millimeter per hour for the ten infants less than 1 month of age, 20.3 Gm. per square millimeter for the sixty-three infants between the ages of 1 month and 1 year and 25.3 Gm. per square millimeter for the infants 1 year of age and more. The mean deviation from the average values in all of the groups of measurements was less than ± 8 per cent.

Until further observations are available, the average figures already mentioned are proposed as tentative basal standards for the insensible perspiration of infants. It must be emphasized that with further investigation, they may require slight revision.

The relatively simple method of estimating the insensible loss by successive weighing has already been utilized for predicting the metabolism of adults by Benedict and Root and that of infants by Levine, Wilson and Kelly. The method appears to be sufficiently accurate for clinical purposes to replace the much more difficult procedure of directly measuring the respiratory exchange of infants. The establishment of normal standards also affords the opportunity of studying certain infantile conditions associated with a probable derangement of water metabolism such as hydrolability (exudative diathesis), nutritional edema and dehydration. Complete studies of the water balance in these conditions are being made at the present time and will be reported at a later date.

THE PREVENTION OF PEMPHIGUS NEONATORUM IN HOSPITALS. DR. ROOD TAYLOR.

Dr. Neff's comprehensive contribution and the discussion aroused by it only one year ago make one hesitate before so soon presenting another paper concerning epidemic pemphigus of the new-born. Consequently, the present paper will be restricted to the description of a method which has been successful in preventing epidemics of this disorder for the past four years and ten months in the nursery for new-born infants of Northwestern Hospital, Minneapolis.

This is a private hospital with 200 beds, with more than 300 obstetric cases annually and with an average of slightly more than 11 new-born infants resident in the nursery. The nursing is done by short term student nurses and by graduate special nurses. The great defect of this method is that the frequent changes in the personnel interfere with a continuous technical routine.

I began my experience as medical supervisor of the nursery for new-born infants in December, 1920. During the next three and one half years, there were two epidemics of pemphigus severe enough to force closing the nursery and the temporary refusal of obstetric cases; in addition, there were a number of minor epidemics which subsided under the scheme of management then in vogue, which included the use of duplicate paraphernalia, two nurseries, two sets of implements, scales, baths, etc., two sets of nurses and the liberal use of ointment of ammoniated mercury, hydrogen peroxide and silver nitrate.

In July, 1924, the technic was changed and the following scheme worked out: On the first day, the infant's skin is oiled; but on the second and succeeding days he is bathed with castile soap and water; the axillary and inguinal spaces are rinsed with boric acid and he is thoroughly dried; then he is powdered liberally with a mixture of mild mercurous chloride, 3 parts, talcum, 2 parts and zinc oxide, 1 part.

Since the use of this technic was begun, there has been no epidemic of pemphigus. In all, seven infants have had pemphigus during the five year period. Two of these were under the care of other physicians and were isolated and treated by them. The remaining five cases were mild, and the children were given no treatment. They were left in the nursery with the other infants, were bathed and cared for by the same nurses and were powdered with the same powder. They received no other treatment. Two of the cases appeared within twenty-four hours of each other; otherwise, during the four years and ten months during which this scheme has been used, there has never been more than one case present in the nursery.

The fact that epidemics have not followed make me believe that these occasional cases have been due to careless drying and inadequate powdering by inexperienced nurses.

This method has certain disadvantages. The cotton underclothing often becomes the color of gray powder. A dermatitis occasionally develops, though it always clears up when a plain talcum powder is substituted.

This dermatitis usually occurs on the lower trunk and anterior thighs in infants who have been circumcised and probably results from the concomitant use of other antiseptics.

With this dry method of treating the infants' skin, the rate of desquamation is slower and consequently the skin feels a little dryer and harsher than it does when oil is used daily. The dry method has the good effect of preventing intertrigo as well as pemphigus.

DISCUSSION

DR. WALTER LESTER CARR: At a previous meeting, Dr. Neff discussed this subject, and I mentioned the difficulty we had had at a maternity hospital in which there were 1,200 or 1,400 confinements a year, and in which we had just been through an epidemic of 65 cases of pemphigus. Ointments and application of powders have been discarded, and the skin is kept dry. An incandescent light of 25 watts is used when necessary to maintain dryness. The results have proved satisfactory, as last year there were only sixteen cases in the institution.

At birth the infants are anointed with sterile albolene or oil and are not bathed. If pemphigus should develop the infant is isolated immediately and is not nursed; the milk is drawn from the mother and the infant is fed from a bottle. When a vesicle is present, the skin is dried by alcohol. The use of other applications has been discontinued, and the infant is handled as little as possible by the nurses.

I believe that in every institution in which there is a change from day to day in the personnel infection is liable to occur, but a simple routine, such as I have indicated, with the use of either sterile albolene or sterile oil without bathing, lessens skin infection. When bathing is done a preparation of castile soap from a shake bottle is preferable to soap in cake form, or soap dipped out of a container. I do not favor the use of antiseptic powder for its antiseptic value.

Formerly nurseries were changed, the walls washed, painting and other things were done, which prevented the everyday use of the rooms. By the present method, the results are better and the nurseries are in continued use.

In most of these epidemics, the smears and cultures are negative.

DR. L. R. DEBUYS: About eight years ago, in my service of the newly born we began to discontinue the initial baths while making observations on the weights and temperatures. These observations were properly controlled, and we found that with the no-bath plan the temperatures were more even, the loss of body weight was less and the return to normal birth weight was more rapid.

We then tried the plan of giving no bath for several days. Now, the routine method in the service is: The infant is immediately oiled as soon as it is brought to the nursery and it is not bathed until it reaches its normal birth weight. A daily oiling, however, is given. We have not had any cases of pemphigus, except on one or two occasions when the infection was traced to private cases, in which the infants were being bathed according to the old routine.

The following story is instructive: A premature infant, a private patient, had received no bath up to the time it was discharged from the institution. At home the mother continued the routine care accorded the infant in the hospital. She made the usual visits to my office, and on the occasion of a visit when the infant was 3 months old the mother asked when she might begin giving it a bath with soap and water. I replied, "You might do so at any time now." I had not remembered on previous visits that the infant had been receiving only its daily oil rub. The point of interest is that the infant had made a particularly creditable progress in weight and well-being and especially that its skin was exceptionally clear and clean.

DR. FRANK C. NEFF, Kansas City, Mo.: I am interested in Dr. Taylor's method of preventing pemphigus. There ought to be some way to make the nursery fool-proof against such a disease. But I think the real reason for the spread is what he mentions. There are outside nurses, outside physicians and other persons who come into the nursery, and it is difficult to standardize any sort of procedure. A routine method that every nurse in the hospital will follow with regard to the care of the skin is worth a good deal of consideration.

I have not tried this particular method, but it sounds as though it might really be of benefit. However, if a hospital is fortunate in having one supervising nurse and one pediatrician who are in complete charge of the rules of the nursery, I think that good results will be obtained, if they are constantly on the job.

DR. WARREN R. SISSON, Boston: In the past few years at the Boston Lying-In Hospital, we have been using the routine methods, taking general care of infants as is the custom in most hospitals. Up to last year or the year before, we were not troubled by any epidemics; a few intermittent cases occurred, but they did not disturb the routine of the hospital.

About two years ago, an epidemic which was almost ubiquitous struck Boston. In every hospital, with possibly one or two exceptions, patients had been infected with various forms of impetiginous pemphigoid lesions. It is striking that without any apparent changes in technic this infection spread through the city, through private as well as public hospitals.

About six or eight months ago, the use of an old but perhaps rejuvenated method was applied; from 4 to 5 per cent ointment of ammoniated mercury was used in all the hospitals in Boston, with few exceptions.

It is interesting that almost coincidentally all of the lesions and all of the incidence of this condition have practically disappeared. It is our custom now for all infants to be washed and immediately given an inunction of from 4 to 5 per cent ointment of ammoniated mercury. After that, the infants are oiled daily until they leave the hospital.

At the present time, no lesions are present in any of the patients.

DR. JOHN A. FOOTE, Washington, D. C.: Cases have been reported of considerable absorption in infants for whom mercury preparations have been used externally, and it would be interesting to know whether Dr. Taylor has made any observation of the rate or degree of absorption of mercury in his series of cases. This could easily be determined from the urine and would have value in enabling one to see whether his method shows a fair or usual or unusual amount of such absorption.

At the Georgetown University Hospital, where there are thirty beds, we have been fortunte in not having an epidemic of pemphigus in the last three years. We have simply used liquid castile soap and a good technic and have watched for lesions in the mother.

The use of some method of antisepsis for the skin which will not be poisonous and will not disfigure the skin by discoloration would be of great value.

DR. ROOD TAYLOR, Minneapolis: I should like to emphasize that I am attempting to describe a method which has worked. It is not necessarily the only method. Moisture tends to increase the growth of bacteria in the skin, and keeping the skin dry is one of the important things. I saw two infants who developed the disease within a few days after leaving the hospital. I believe they picked up the infection in the hospital, but the way the skin was treated there inhibited the disease from appearing. There was no examination of the urine for mercury. There was no evidence of the action of mild mercurous chloride.

Second Session, May 20, 8:30 p. m.

DIET AND DENTAL CARIES. DR. PHILIP C. JEANS and DR. JULIAN D. BOYD.

We have observed the arrest of dental caries in many children under divergent conditions of health and environment subsequent to the use of controlled diets. The softened dentine became normally hard after about ten weeks of dietary supervision, and the caries showed no progress as long as the children adhered to the prescribed diets. Children who reverted to their previous habits of eating had a recurrence of active caries. The response to diet has been observed in healthy children in their home environment, and in various groups of patients in

the hospital. One group of patients had diabetes mellitus, another had celiac disease and the remainder had orthopedic conditions.

The diets of the healthy children and of those with diabetes mellitus included a daily minimum allowance of 1 quart of milk, one egg, 1 teaspoonful of cod liver oil, 1 ounce of butter, one orange, and two or more servings of succulent vegetables and of fruits, supplemented with other foods to supply adequate protein and calories. The nature of the other foods was not restricted in the case of the normal children; the diabetic patients received limited amounts of carbohydrate, and most of their calories were supplied by fat. The diets of children with celiac disease necessarily were limited in fat and in fiber. They received a quart of skimmed milk, no butter, and no fibrous or starchy vegetables or fruits. With these modifications, their diets included the minimum factors already enumerated. Their calories were supplied chiefly by dextrose.

Arrest of caries resulted with diets high in sugar in one group, and with the carbohydrate restricted to a minimum, in another group. It followed the use of diets high in fat as well as those which were practically fat free. One common factor of all diets was a high content of vitamins and minerals. Studies of salivary acidity and of the potential acid producing ability of the peridental flora of those with arrested, as compared with those with active, caries have shown no noteworthy difference between the two groups. Mouth hygiene did not appear to be a factor.

In addition to the arrest of caries, the children have shown evidences of better health coincident with the dietary supervision. On the basis of the clinical and experimental observations, it is thought that active caries may well be viewed as indicative of dietary deficiencies which may not make themselves obviously apparent in any other way.

Effect of Rickets and Severe Nutritional Disturbances on Children's Teeth. Dr. Frank C. Neff.

Experimentally, the effect of diet on the teeth of animals has been clearly shown by Mellanby and others. In children diet is only one of the factors, not so important as the nutritional disturbance, usually rickets, which has an unquestioned influence on the mineral structure of the teeth. Long continued severe infections and undernutrition beginning early in life and continuing through childhood sometimes cause changes in one or both sets of teeth.

Three evidences of disturbance are found: delayed eruption of the teeth, often of little significance except that it may have to do with a retardation in calcification; gross defects and deformities, especially in the enamel, with markings such as pitting, thinness of the enamel, superimposed growths, ridges, furrows and bands around the teeth; microscopic and chemical evidences of imperfect calcification of the structure of the dentine and enamel.

I have attempted to determine whether during active nutritional disturbance, especially accompanied by negative mineral balance, the teeth will show roentgen evidence of insufficient calcification. Serial films during appropriate treatment may permit one to see an improvement in the density of the unerupted calcifying teeth.

Discussion on Papers of Drs. Jeans and Boyd and Dr. Neff

Dr. James L. Gamble, Brookline, Mass.: As well as I can recall from the experiments of Wolbach and Howe, vitamin C was indicated as indispensable for the solidification of dentine. I should like to ask Dr. Boyd whether all of the diets, by means of which he obtained arrest of caries, contained orange juice.

Dr. Henry J. Gerstenberger, Cleveland: I have had occasion to feed after birth and over long periods children, especially members of my own family, all the essential food constituents in sufficient quantities, including cod liver oil and orange juice, and I have not been able to prevent the development of small areas of dental caries in some cases. Occasionally, infections occurred in these children, and they may have been the cause of the caries either directly or indirectly

through loss of appetite. I am certain that the amount of orange juice and cod liver oil always was ample, as was the intake of proteins and minerals. Consequently, I have felt that in the production of some of the cases of caries a factor other than the one of proper and adequate food plays a rôle.

DR. P. C. JEANS, Iowa City: One point mentioned in the paper should be emphasized. The activity of dental caries is determined by factors operating at the time of observation rather than by anything which happened in the remote past. Teeth may be malformed because of former rickets or for other reasons and thus may be more susceptible to decay than sound teeth, but they will not decay if the diet is adequate. If decay has begun, it can be arrested in the course of a few weeks by means of a proper diet. Though these statements are based on a relatively small number of cases, no exception has been encountered. In every instance in which the children were placed on a supervised dietary regimen, caries has become arrested. In several of the children, the caries became arrested in spite of infections which were present. Thus, we have come to believe that the presence or absence of active caries depends wholly on the nature of the diet.

DR. J. H. MASON KNOX, JR., Baltimore: Dr. Jeans answered the question that I had in mind.

We all recognize the value of adequate diet in diminishing the amount of dental caries in young children. I hope the striking results obtained in this way by Dr. Jeans and Dr. Boyd can be generally confirmed. I have heretofore considered that the quality of the dental structure was partly influenced by inheritance. I have repeatedly seen children in the same family and presumably fed the same kind of food differ greatly among themselves as to the amount of dental caries. In a number of these families the quality of the teeth in the parents differed, and I had inferred that the teeth of the children may have resembled those of one or the other parent.

I should like to ask the writers whether the character of the teeth of the parents or near relatives was marked in their cases.

In examining groups of children, I have noticed also marked average differences in the amount of dental caries in various communities, with no obvious differences in the diet to explain it.

I have been struck, too, on several occasions with the greater prevalence of dental caries among white as compared to negro children, although the usual rachitic bony deformities were about twice as common among the latter group.

DR. J. D. BOYD: In each of the groups we observed, all the children received orange juice every day.

It is not correct to state that proper diet is the only factor in the arrest of caries. However, in every instance in which we have been able to control the diet, the caries has become arrested. The diet of a child may be less adequate than is apparent from the nature of the daily menu as stated by the parent. We have had parents assure us that their children were receiving diets which were essentially identical with that which we prescribed; yet, when they kept careful record of the amounts which the children ate of the foods offered them, the intake was not representative of a well balanced menu. When the prescribed diet was actually ingested, the caries became arrested and the children gained in weight.

It has been reported that in dental clinics where a large foreign element is examined, not only the children but also the adults will have excellent teeth when they first come to this country. Within a short time after they have assumed the food habits of their new environment, the teeth develop extensive caries.

We have not had an opportunity to correlate the condition of the children's teeth with that of their parents. We feel that probably familial food habits are more significant than hereditary factors in the case of whole families showing extensive caries.

In the orthopedic group, two of the children remained in the clinic for some time after the completion of the experiment. It was impossible to continue close dietary supervision, and they resumed their former eating habits, choosing from

the foods offered them those which they liked the best. Reexamination after a period of several months revealed reactivation of caries.

We purposely left cavities unfilled in the teeth of the children in whom caries had been arrested. The cavities were of such a nature that food débris was necessarily present; yet repeated examinations over periods of months offered no evidence of progression of the caries.

DR. FRANK C. NEFF: In conclusion, I wish to emphasize a thing which it seems to me might have some value in the care of children's teeth. By roentgen examination of the unerupted teeth in children one can determine whether calcification is going ahead properly and whether what is being done for the child in the way of diet and treatment is having any beneficial effect.

COMPOSITION OF BONE: VI. EFFECT OF MASSIVE DOSES OF IRRADIATED ERGOSTEROL. DR. BENJAMIN KRAMER, M. J. SHEAR and MARGARET R. MCKENZIE.

Klein (Effects of Massive Doses of Irradiated Ergosterol, *J. A. M. A.* **92**:621 [Feb. 23] 1929) recently reported experiments in which massive doses of irradiated ergosterol were fed to young rats. The three groups of rats all received Steenbock's ration 2965. One group served as a control. The second group received 2 Gm. of cod liver oil to every 100 Gm. of the ration, while the third group received massive doses of irradiated ergosterol in addition to the ration. The irradiated ergosterol was obtained through the courtesy of Dr. Cottrell of the Winthrop Chemical Company, Inc., New York.

At the end of the experimental period of one month, autopsies were performed on the rats, and the bones were placed at our disposal for analysis. The present paper is a report of the analyses of these bones.

The serum calcium of the control rats was 10.9 mg. per hundred cubic centimeters; that of the group receiving cod liver oil was 13.9 mg. per cent and that of the group fed irradiated ergosterol was 16.1 mg. per cent. It seemed possible that this 50 per cent increase in the serum calcium of the rats which had received irradiated ergosterol might have affected the ratio $\frac{\text{residual Ca}}{\text{P}}$ in the bones of these rats. The bones were therefore prepared and analyzed according to the method of Shear and Kramer (*J. Biol. Chem.* **79**:105, 1928). There was one notable difference in technic; phosphorus was determined by the method of Fiske and Subbarow (*J. Biol. Chem.* **66**:375, 1925) instead of by the Briggs-Bell-Doisy method.

The mean of all the ratios obtained was 2.00, with an average deviation of $\pm$ 0.05. This compares well with the value previously obtained for the bones of normal rat by Kramer and Shear (*J. Biol. Chem.* **79**:147, 1928) in which a mean of 1.99 was found with an average deviation of $\pm$ 0.05. For the control rats, ratios of 2.09 and 2.02 were obtained; for the rats which had received irradiated ergosterol, the ratios obtained were 2.03, 2.00, and 2.04. There is thus no discernible difference between these two groups. For the group given cod liver oil ratios of 1.91 and 1.93 were obtained. Although the ratios of the group receiving cod liver oil are both lower than those of the other two groups, we do not at present attach significance to these differences because they are within the range of reproducibility of the microtechnic.

THE ANTIRACHITIC FACTOR CONTENT OF HUMAN MILK: A REPORT ON EFFORTS TO INCREASE IT. DR. H. J. GERSTENBERGER, DR. J. I. HARTMAN, DR. G. R. RUSSELL, DONALDA N. SMITH and DR. HELEN BRONSON BROWN.

The following methods were used in the attempt to make human milk produced by wet nurses sufficiently antirachitic to cure human infantile rickets:

A. The daily ingestion by wet nurses of 1 tablespoonful of cod liver oil which had been proved to be potent in curing infants with rickets when fed to them in amounts varying from 2.2 to 3.5 cc. in certain foods.

B. The daily exposures of the bodies of wet nurses to the quartz lamp, alternating front and back.

C. The daily exposure of the face, neck, arms and chest triangles of the wet nurses to the summer sun while seated facing it on top of the six story hospital building from 11 to 11:45 a. m.

D. The daily ingestion of 6 Gm. of cholesterol, irradiated with the quartz lamp by Dr. Harry Goldblatt of the Department of Pathology of Western Reserve University.

E. The daily ingestion of from 10 to 15 mg. of irradiated ergosterol, with the precaution of weekly determinations of the blood serum calcium and inorganic phosphate levels of the wet nurses.

The following results were obtained:

1. Method A was of no value in three rachitic infants after fifty-one, sixty-three and sixty-eight days of observation at the begininng of which the respective infants received the human milk for thirty-three, forty-five and thirty-six days. Subsequent feeding of foods containing from 3.5 to 2.2 cc. of cod liver oil was followed by a satisfactory return of the bones and blood to normal conditions.

2. Method B gave to the milk definite antirachitic value. However, the healing produced in the two rachitic infants observed was not as rapid as that obtained by feeding rachitic infants foods containing from 3.5 to 2.2 cc. of cod liver oil.

3. Method C was effective in transmitting antirachitic power to the milk of the wet nurses. However, the potency of this human milk was decidedly less than that developed by method B. Of the three rachitic infants fed this milk, one showed only the slightest beginning of healing on the sixtieth day. After taking wet nurses' milk for thirty-six days before the producers had been exposed regularly to the sun, a second infant showed slight healing which changed but little until the infant took, for thirty days, milk produced by the wet nurses after they were regularly exposed to sunlight. At this point healing increased, but even so, it was slow, reaching not more than the 40 per cent healing stage at the end of eighty-five days. During this period, spasmophilia (clinical and blood) developed. A third infant showed beginning improvement with the forty-second day and only slightly more healing on the fifty-seventh day, when the observation ended.

4. Method D did not give to the milk of the wet nurses any demonstrable antirachitic value as observed by feeding it to one rachitic infant for sixty-seven days and to another for fifty-seven days. Both infants responded promptly, but somewhat slowly, to weekly exposures of artificially produced ultraviolet rays.

5. Method E was highly successful, the one infant with mild rickets showing beginning healing after twelve days and complete healing after forty-six days and the other with severe rickets showing definite healing on the fifteenth day and practically complete healing on the fiftieth day.

The blood serum calcium and inorganic phosphate levels of the wet nurses remained within normal limits and varied from week to week only slightly. Sixteen weekly checks were made on the blood of the first wet nurse, six on the second and eight on the third. Determinations of blood pressure and examinations of the urine likewise gave negative results. Nevertheless, at this time, we do not wish to recommend for general application the doses of irradiated ergosterol that we have used in this work.

DISCUSSION

DR. JULIUS H. HESS: Did I understand Dr. Gerstenberger to say that exposure of the mother to the direct sun ray did not influence the antirachitic factor in her breast milk?

DR. H. J. GERSTENBERGER: In answer to Dr. Hess' question regarding the method used to expose the wet nurses to sunlight, I may repeat that the women were exposed at the top of the six-story hospital building during the summer from 11 to 11:45 a. m., with the face, arms and chest triangle exposed. If the

entire integument of the wet nurses had been irradiated, I am certain that milk with greater antirachitic powers could have been obtained. We were interested, however, in getting an idea as to the antirachitic value of milk produced by American mothers who were in all probability exposed as much and maybe more than an average American mother is as she lives and works and plays during the summer.

On the basis of the observations, it is clear that human milk produced by human mothers cannot have any antirachitic qualities of practical value during the winter unless the skin of the mother is adequately exposed to artifically produced ultraviolet rays or unless they are ingesting irradiated ergosterol in sufficient quantities.

THE TREATMENT OF CONSTIPATION BY THE USE OF A RESIDUE-FREE DIET. DR. RICHARD M. SMITH and DR. HENRY E. GALLUP.

Severe constipation is one of the common conditions encountered in pediatric practice. By constipation is meant not that a child occasionally misses a regular bowel movement, but that movements occur only with the use of vigorous catharsis and enemas. Children with chronic constipation frequently suffer from attacks of abdominal pain associated with vomiting, fever and "acidosis," presenting the picture of malnutrition. They tire easily.

Treatment with a diet containing a large roughage residue not infrequently fails to correct the condition. In many instances, an abnormal or a disturbed emptying of the colon, due to redundance or lack of tone, is the underlying cause of the constipation. This abnormal function is understood more clearly if the behavior of the colon is studied under the fluoroscope during the injection of a barium enema. This study reveals masses of hard fecal material in the colon and adherent to the wall. The colon is redundant, overdistended, and frequently lacking in tone. Evacuation is incomplete. Children presenting this picture may be successfully treated by the administration of a diet containing a minimum amount of residue. This is accomplished by straining all food which has any cellulose content. Mineral oil should also be given in small doses after each meal. Catharsis may be necessary in the preliminary stages of the treatment in the form of castor oil weekly. A daily enema at night will produce a movement. This enema should be given with the bag never more than 18 inches above the buttocks, with the patient lying down, and not more than 8 or 10 ounces (236 or 295.7 cc.) of liquid used.

As the condition improves, enemas and catharsis may be eliminated, the diet gradually increased in residue content, and finally mineral oil omitted. Illustrative cases are presented.

DISCUSSION

DR. P. C. JEANS: Will Dr. Smith give a little more detail concerning the constituents of the diet?

DR. ROOD TAYLOR: Do I understand, Dr. Smith, that you believe the colon should be completely emptied every day?

DR. RICHARD M. SMITH: First, as to the constituents of the diet, the children are given a full diet in every respect except that an attempt is made to eliminate all the residue by straining. All vegetables are boiled and strained, and the most fibrous vegetables, such as celery, are not given at all. In the severe cases, orange juice is strained in order to avoid any possible residue.

Of course, the colon is not entirely emptied every day, but little fecal material is retained from one day to the next.

THE RELATION OF WEIGHT, HEIGHT AND AGE IN NEGRO CHILDREN. DR. L. T. ROYSTER and C. N. HULVEY.

This paper will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

Book Reviews

DISEASES AND DEFORMITIES OF THE SPINE AND THORAX. By ARTHUR STEINDLER, M.D., Professor and Head of the Department of Orthopedic Surgery of Iowa State University Medical School, Iowa City. Cloth bound. Price, $12. Pp. 573, with 76 plates. St. Louis: C. V. Mosby Company, 1929.

The development of the topic, dealing broadly with basic, theoretical considerations and experimental evidence has caused this book to be different from the usual plan of books for a similar purpose.

An attempt is made to develop orthopedic judgment, so as to apply the proper decision to a given problem.

The seventy-six plates are evenly distributed throughout the text, and each plate is made up of a group of photographs, x-ray prints and diagrams illustrative of the adjacent subject matter of the text.

At the end of each chapter there is a résumé and a comprehensive bibliography.

Chapter 1 deals with the congenital deformities, which are determined in a coherent and systematic manner by powerful early developmental nature.

The static constitutional anteroposterior deformities of the spine are dependent on a proper relationship between the spine, the thorax and the abdominal cavity. Any change in one of the systems must be accompanied by changes in the others.

The treatment of scoliosis involves mobilization, correction and maintenance. This is accomplished by various means, and each case requires separate consideration and treatment.

Fractures and dislocations produce deformities, which in themselves are not of primary consideration. The dislocations of the cervical spine which are successfully reduced are the exception, because of late diagnosis and because they rapidly become fixed and are unreducible.

The conservative treatment is the one of choice except in cases with complications, when operative measures are indicated.

In cases of pain in the lower part of the back, one must distinguish between ligamentous sprain and permanent relaxations, because in the former the condition readily improves, while in the latter operative fusion is required.

The secondary sciatic symptoms should yield to immobilization. Errors of treatment usually result from inefficient immobilization and from weight-bearing too soon after trauma.

Tuberculosis of the spine must be considered as a local manifestation of a generalized infection. In children the cure is accomplished earlier by a bony back, while in adults the end-result is scar tissue, which is less reliable as far as cure is concerned.

Weight-bearing is the worst factor, and this can be eliminated only by recumbency. The cold abscesses will spontaneously disappear under efficient care. In most cases operative fixation has greater indications for the ambulatory cases.

Compression myelitis resulting in paraplegia yields to recumbency in children in most cases, while in adults there are more exceptions.

Osteomyelitis of the spine requires early drainage, which is readily accomplished in most cases, as the arches are involved as a rule. However, if the pathologic process is in the bodies of the vertebrae, the procedure is more difficult.

Syphilitic spondylitis is much more common than is usually expected. Hereditary cases are often associated with involvement of other joints, and the relatively rapid recovery under antisyphilitic therapy is often the best diagnostic test.

The tabetic arthropathies are not rare, but are not correctly diagnosed. In these cases the spine requires general medication and support.

At the beginning of each chapter, there is a well arranged outline of the material, and throughout the text these subheads are distinctly printed in large type.

This book is too extensive for the use of the average medical student but should be good for special reference.

The author has accomplished his desire in writing this text, in that it will stimulate thought on the part of the reader. He has not set forth any dogmatic ideas, and yet has not maintained the beaten path, which is followed by most authors.

The book will be interesting reading to orthopedic surgeons, and to all others who wish to investigate orthopedic conditions of the spine and thorax.

NOTES ON CHRONIC OTORRHOEA. By A. R. FRIEL, M.A., M.D., F.R.C.S. Price, $2.25. Pp. 87, with illustrations. New York: William Wood & Company, 1929.

This book, consisting of eighty-seven pages, many of which are taken up with diagrams and illustrations of the equipment used, is brief and concise. Its brevity is made possible by the absence of unnecessary language. The first twenty-four pages are taken up in an explanation, by simple experiments, of the physics and chemistry of zinc ionization, which is the basis of the author's treatment. The next forty pages deal with the technic of treatment, which includes methods of examination and diagnosis. The remaining portion gives tables of statistics on results of treatment and hints on the organization of the clinic.

He divides his cases of chronic otorrhea into four groups: (1) accessible sepsis, e.g., tympanic sepsis; (2) accessible sepsis with a second factor in the ear, e.g., polypus; (3) accessible sepsis with inflammation in an adjacent organ, e.g., rhinitis, and (4) inaccessible sepsis: (*a*) accessible with difficulty, e.g., cell in mastoid opening directly into tympanum; (*b*) totally inaccessible, chronic mastoiditis.

Conclusions on statistics reported by Dr. T. B. Jobson are: 1. Zinc ionization will cure any case of chronic otorrhea which is curable by drops. 2. It will accomplish this in one-hundredth part of time, thus saving an enormous amount of the patient's, the doctor's and the hospital's time. 3. It will cure a large number of cases which do not mend with ordinary antiseptic treatment.

THE NEWBORN INFANT. By EMERSON L. STONE. Price, $2. Pp. 183. Philadelphia: Lea & Febiger, 1929.

This small volume on the new-born infant gives a lucid résumé of the problems which confront the physician during this age period. Certain obstetric phases of the subject are clearly explained, especially those dealing with the treatment for asphyxia, in which the common violent resuscitative measures are condemned, and those describing the conditions growing out of the fad for routine analgesia. The chapter on "birth injuries" is unusually complete and well written for the size of the book. The fact that the author is an obstetrician offers encouragement to those interested in this much neglected field, for it is to the obstetrician that one must look for aid. The volume will probably be of much more value to the physician who practices obstetrics than to the pediatrician. Its perusal only goes to emphasize more than ever the lack of definite knowledge about the new-born period.

STATIONS THERMALES DU CENTRE DE LA FRANCE. By PAUL CARNOT and others. Price, 12 francs. Pp. 80, with 1 map. Paris: L'Expansion Scientifique Francaise, 1929.

This book is a concise description of the mineral springs of central France, by members of the Hydrotherapy Society of Paris. Each district is briefly described as to situation, climate, composition of the water, physiologic action and the technic of its use in medication.

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INTERNATIONAL CONGRESS ON PEDIATRICS

Prof. I. Jundell, Artillerigatan 23, Stockholm, Sweden.
Place: Stockholm. Time: Aug. 17-20, 1930.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON DISEASES OF CHILDREN

Chairman: Dr. William Weston, 1231 Bull St., Columbia, S. C.
Secretary: Dr. C. A. Aldrich, 545 Lincoln Ave., Winnetka, Ill.
Place: Detroit. Time: 1930.

AMERICAN PEDIATRIC SOCIETY

President: Dr. Joseph Brennemann, 30 N. Michigan Blvd., Chicago.
Secretary-Treasurer: Dr. Howard Childs Carpenter, 1805 Spruce St., Philadelphia.
Place: Montreal. Time: May or June, 1930.

THE ASSOCIATION OF AMERICAN TEACHERS OF THE DISEASES OF CHILDREN

President: Dr. Lawrence T. Royster, State Board of Health, University, Va.
Secretary: Dr. Ralph M. Tyson, 334 S. 21st St., Philadelphia.

AMERICAN CHILD HEALTH ASSOCIATION

President: Hon. Herbert Hoover, 370 7th Ave., New York.
Secretary: Dr. Philip Van Ingen, 370 7th Ave., New York.
Place: Minneapolis. Time: Sept. 30-Oct. 4, 1929.

CANADIAN SOCIETY FOR THE STUDY OF DISEASES OF CHILDREN

President: Dr. Thomas Crossan Clark, 51 Herkimer St., Hamilton, Ont.
Secretary-Treasurer: Dr. Frank H. Boone, 164 James St. S., Hamilton, Ont.

SECTIONAL

CENTRAL STATES PEDIATRIC SOCIETY

President: Dr. J. Gurney Taylor, 120 E. Wisconsin St., Milwaukee.
Secretary: Dr. A. H. Parmelee, 310 S. Michigan Ave., Chicago.
Place: Milwaukee. Time: Sept. 27-28, 1929.

SECTION ON PEDIATRICS OF SOUTHERN MEDICAL ASSOCIATION

Chairman: Dr. J. H. Eugene Rosamond, 1074 Madison Ave., Memphis, Tenn.
Secretary: Dr. A. J. Waring, De Renne Apts., Savannah, Ga.
Place: Miami, Fla. Time: Nov. 20-22, 1929.

LOCAL

ALABAMA PEDIATRIC SOCIETY

President: Dr. J. MacBell, Mobile.
Secretary-Treasurer: Dr. Hughes Kennedy, Jr., Highland Plaza Apts., Birmingham.

NORTHERN CALIFORNIA PEDIATRIC SOCIETY

President: Dr. Edward B. Shaw, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. Lloyd Hardgrave, 384 Post St., San Francisco.
Place: Various teaching schools. Time: Monthly.

SOUTHWESTERN PEDIATRIC SOCIETY

President: Dr. Ezra S. Fish, Pacific Mutual Bldg., Los Angeles.
Secretary: Dr. Fred L. Glascock, 1136 W. 6th St., Los Angeles.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

THE ROCKY MOUNTAIN PEDIATRIC SOCIETY

President: Dr. Elsie Seelye Pratt, Republic Bldg., Denver.
Secretary-Treasurer: Dr. Wilford W. Barber, Metropolitan Bldg., Denver.
Place: Children's Hospital, Denver. Time: Third Saturday of each month from September to May, inclusive, 2:30 p. m.

GEORGIA PEDIATRIC SOCIETY

President: Dr. W. N. Adkins, Medical Arts Bldg., Atlanta.
Secretary-Treasurer: Dr. E. N. Gleaton, 212 E. Gaston St., Savannah.

PEDIATRIC SECTION OF FULTON COUNTY MEDICAL SOCIETY, ATLANTA, GA.

Chairman: Dr. Lee Bivings, Exchange Bldg., Atlanta.
Secretary: Dr. William Kiser, Medical Arts Bldg., Atlanta.
Place: Academy of Medicine, 32 Howard St. Time: Second Thursday of each month from October to April, 8 p. m.

NEW ENGLAND PEDIATRIC SOCIETY

President: Dr. Edwin T. Wyman, 483 Beacon St., Boston.
Secretary-Treasurer: Dr. Randolph K. Byers, 66 Commonwealth Ave., Boston.
Place: Boston Medical Library. Time: Second Friday of each month.

CHICAGO PEDIATRIC SOCIETY

President: Dr. Arthur H. Parmalee, 310 S. Michigan Blvd., Chicago.
Secretary: Dr. Bert I. Beverly, 130 S. Oak Park Ave., Oak Park, Ill.
Place: City Club. Time: Third Tuesday of each month.

LOUISIANA STATE PEDIATRIC SOCIETY

President: Dr. J. A. Crawford, Lake Charles.
Secretary-Treasurer: Dr. C. T. Williams, 7401 Burthe St., New Orleans.

DETROIT PEDIATRIC SOCIETY

President: Dr. M. Boyd Kay, 10 Peterboro St., Detroit.
Secretary: Dr. Charles A. Wilson, 71 E. Ferry St., Detroit.
Place: Children's Hospital of Michigan. Time: No fixed date; approximately once a month.

NORTHWESTERN PEDIATRIC SOCIETY

President: Dr. C. R. Moriarity, La Salle Bldg., Minneapolis.
Secretary: Dr. E. S. Platou, 730 La Salle Bldg., Minneapolis.
Place: University Campus and Rochester, Minn. Time: Winter, Spring, Summer, Fall.

ST. LOUIS PEDIATRIC SOCIETY

President: Dr. Park J. White, Beaumont Bldg., St. Louis.
Secretary-Treasurer: Dr. Ralph L. Cook, 508 N. Grand Blvd., St. Louis.
Place: St. Louis Children's Hospital. Time: At least once a year and contingent thereafter on the arrangement of a suitable program.

NEBRASKA-IOWA PEDIATRIC SOCIETY

President: Dr. Floyd Clarke, City Nat'l Bank Bldg., Omaha.
Secretary: Dr. R. B. Eldredge, Medical Arts Bldg., Omaha.
Place: Add-Sell Restaurant. Time: Second Thursday of each month from October to May, inclusive, 6 p. m.

BROOKLYN PEDIATRIC SOCIETY

President: Dr. Louis M. Ruderman, 961 Eastern Pkwy., Brooklyn.
Secretary: Dr. Joseph F. Paulonis, 1357 Bushwick Ave., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Last Wednesday of each month, except June, July and August.

BRONX PEDIATRIC SOCIETY

President: Dr. L. H. Barenberg, 1749 Grand Concourse, New York.
Secretary: Dr. Joseph Golomb, 496 E. 175th St., New York.
Place: Concourse Plaza, 161st St. and Grand Concourse. Time: Second Wednesday of each month, except June, July, August and September.

CENTRAL NEW YORK PEDIATRIC CLUB

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various cities in New York. Time: Fourth Thursday of October, January and April.

NEW YORK ACADEMY OF MEDICINE, SECTION OF PEDIATRICS

President: Dr. F. Elmer Johnson, 300 West End Ave., New York.
Secretary: Dr. Hugh Chaplin, 39 E. 75th St., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

ROCHESTER PEDIATRIC SOCIETY

President: Dr. John Aikman, 184 Alexander St., Rochester, N. Y.
Secretary: Dr. Howard F. Rowley, 278 Alexander St., Rochester, N. Y.
Place: Arranged by program committee. Time: First Friday of each month from October to May.

ACADEMY OF MEDICINE OF CLEVELAND, PEDIATRIC SECTION

Chairman: Dr. C. W. Wyckoff, 8314 Euclid Ave., Cleveland.
Secretary: Dr. C. L. Ruggles, 10616 Euclid Ave., Cleveland.
Place: 2009 Adelbert Road. Time: Four meetings, from September to May.

OKLAHOMA STATE PEDIATRIC SOCIETY

President: Dr. William M. Taylor, Osler Bldg., Oklahoma City.
Secretary: Dr. Carol Pounders, 110 W. 10th St., Oklahoma City.
Place: Shawnee. Time: May, 1930.

PHILADELPHIA PEDIATRIC SOCIETY

President: Dr. Ralph M. Tyson, 334 S. 21st St., Philadelphia.
Secretary: Dr. John D. Donnelly, 115 Bryn Mawr Ave., Bala-Cynwyd, Pa.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday of each month from October to June, inclusive.

PITTSBURGH PEDIATRIC SOCIETY

President: Dr. Zaccheus R. Scott, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. Theodore C. Elterich, Highland Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate months.

THE SOUTH CAROLINA PEDIATRIC SOCIETY

President: Dr. E. A. Hines, Seneca.
Secretary-Treasurer: Dr. R. M. Pollitzer, 105 E. North St., Greenville.

MEMPHIS PEDIATRIC SOCIETY

President: Dr. A. G. Quinn, 1193 Madison Ave., Memphis, Tenn.
Secretary: Dr. Kinsey M. Buck, 1024 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month.

TEXAS PEDIATRIC SOCIETY

President: Dr. H. P. Ledford, 1310, 9th St., Wichita.
Secretary-Treasurer: Dr. John G. Young, 3930 McKinney Ave., Dallas.

RICHMOND PEDIATRIC SOCIETY

President: Dr. J. B. Stone, 2042 Park Ave., Medical Arts Bldg., Richmond, Va.
Secretary-Treasurer: Dr. Samuel A. Anderson, Jr., 2326 W. Grace St., Richmond, Va.
Place: Westmoreland Club, 601 E. Grace St. Time: First Thursday of each month (other than during the summer).

VIRGINIA PEDIATRIC SOCIETY

President: Dr. Charles E. Conrad, Harrisonburg.
Secretary: Dr. William B. McIlwaine, Petersburg.

NORTH PACIFIC PEDIATRIC SOCIETY

President: Dr. E. D. Carder, 736 Granville St., Vancouver, B. C.
Secretary-Treasurer: Dr. George A. Lamont, 736 Granville St., Vancouver, B. C.

SEATTLE PEDIATRIC SOCIETY

President: Dr. Arthur H. Gray, Lumber Exchange Bldg., Seattle.
Secretary: Dr. Armin Rembe, Stimson Bldg., Seattle.
Place: College Club. Time: Third Friday of each month, 7 p. m.

MILWAUKEE PEDIATRIC SOCIETY

President: Dr. A. L. Kastner, 120 E. Wisconsin St., Milwaukee.
Secretary: Dr. F. R. Janney, 418 E. North Ave., Milwaukee.
Place: Children's Hospital. Time: Second Wednesday of each alternate month, beginning with February.

ACADEMY OF MEDICINE, TORONTO, SECTION OF PEDIATRICS

President: Dr. G. A. Davis, 12 Belsize Drive, Toronto.
Secretary: Dr. Gladys Boyd, 56 DeLisle St., Toronto.
Time: November 17, December 15, February 16 and March 15.

THE AMERICAN JOURNAL OF DISEASES OF CHILDREN is published by the American Medical Association as a means for the advancement of knowledge of the diseases of children and for the publication of scientific investigation in this field.

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